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SYPHILIS OF THE LUNG:

WITH A REPORT OF THE ANATOMICAL FINDINGS IN A CASE.

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Syphilis of the lung is either congenital or acquired. In this paper only the latter form will be considered.

Whether we agree with the majority who hold that syphilis of the lung is rare in the adult, or with the few who consider it more common, we all must accept the opinion of the best authorities—that the condition does exist. Now, when it is taken into consideration that syphilis is curable and that the advanced cases of pulmonary tuberculosis, with which it is likely to be confounded, are, as a rule, incurable, one should be all the more careful to make no mistakes.

That syphilis of the lung is a very rare condition is almost universally conceded by men of large experience. Osler states that in twenty-five years he has not seen more than half a dozen specimens in which there was no question as to the nature of the trouble.[1] In 1898 the London hospitals and Royal College of Surgeons contained only ten specimens which seemed to be unquestionable examples of this disease.[2] Among 13,000 specimens in the Army Medical Museum at Washington there is not one of this disease.

There are others, however, who hold a contrary opinion. Porter considers it common in adults. Satterthwaite[3] thinks that lung syphilis is greatly underestimated by the general practitioner, and "almost unknown to many syphilographers." Pankritius, Schnitzler, and Grandidier have also found it by no means unusual.

[1] Practice of Medicine. [2] Fowler and Godlee. The Diseases of the Lungs.
[3] Boston Medical and Surgical Journal, 1891, vol. cxxiv.

Stengel[1] suggests that this difference of opinion depends largely upon the point of view of the observer—whether as a clinician or as a pathologist, the former believing that this condition is comparatively common, the latter that it is very unusual. It must be acknowledged that the pathologist has by far the best of it when it comes to producing convincing evidence. There is no disease, however, in which it is more necessary to carefully weigh the pathological findings with the clinical history before giving an opinion.

Without questioning their rarity, it may be well to mention one or two possible factors which have tended to reduce the number of cases reported. In the first place, it is most difficult to differentiate between pulmonary tuberculosis and pulmonary syphilis. This is well recognized, not only at the bedside, but in the pathological laboratory. In fact, the only absolutely certain point of difference in many cases is the presence of the tubercle bacillus, and even this unquestionable evidence of tuberculosis does not preclude the possibility of associated syphilis. Pulmonary tuberculosis is so common and syphilis of the lung thought to be so unusual that it seems probable that some of these cases, because of their very rarity, may slip by with a diagnosis of tuberculosis. Many cases of supposed phthisis give such a typical history and show such characteristic signs of the disease that it does not seem necessary to examine the sputum, or, even if the bacilli are not found after a few examinations, no change in the diagnosis seems called for. Satterthwaite[2] gives a very interesting example of such a case which fell into his hands and was subsequently cured by appropriate treatment.

The same line of argument is used in the post-mortem room. The condition is so much more likely to be tuberculous than luetic that it is almost always placed in that class unless pronounced syphilitic lesions of other organs are discovered.

Tertiary Lesions. While secondary lesions of the trachea have been occasionally observed,[3] there are, so far as I know, no records of such lesions in the bronchi. From the similarity of structure and function, however, there seems to be no reason why similar secondaries (mucous patches) might not be found, should an opportunity for their observation offer. The results of antisyphilitic treatment upon bronchitis occurring during secondary manifestations also serves to strengthen the theory of possible secondary bronchial involvement.

Tertiary lesions of the trachea and bronchi, consisting of gummatous infiltration of the submucous tissue, with consequent fibrous thickening and stenosis, are not unusual, and the lesions of the pulmonary parenchyma are solely of the tertiary type.

The Period Intervening between the Primary Infection and the Appearance of Pulmonary Lesions. Undoubted syphil-

[1] University of Pennsylvania Medical Bulletin, 1903–1904.
[2] Loc. cit.
[3] Morrell MacKenzie. Diseases of the Throat and Nose, London, 1880, vol. i. p. 510.

itic disease in the lungs has been recorded as early as one and as late as twenty years after the initial sore. The most usual time is, however, from the fifth to the tenth year after the infection. Frank[1] and Schirren[2] have reported cases only a few months after infection. My own case showed comparatively recent lesions twenty-one years after infection.

THE LESIONS OF SYPHILIS OF THE LUNG. 1. Gumma. 2. Pneumonia. 3. Fibrosis.

1. Gummata are either single or multiple, round, elliptical, or irregular in shape, and vary in size from a hemp-seed to a goose-egg, and in consistency from a soft colloid to a firm fibroid or cheesy tumor.

They may be found in any part of either lung, but are more usual within the substance and in the vicinity of the root, though they may be at the apex or upon the surface (my own case). Many observers claim that the right middle lobe is the favorite location. Grandidier[3] reported 30 cases in which the right middle lobe was the seat in 27. Hirsch[4] quotes Pankritius' statistics of 105 cases, in which 76 were right-sided, 18 left-sided, and 11 bilateral.

A gumma may present any of the following appearances, depending somewhat upon its age: It may be a gelatinous mass or a nodule of a grayish-white or yellow or reddish color, usually surrounded by a more or less distinct capsule, formed through inflammatory changes, set up in the surrounding lung tissue. On section there is a tendency for the centre to bulge somewhat, which is considered to be of some value as a distinction from tubercle, there being apparently more elasticity in the periphery of the gumma.

Microscopically they are found, like gummata of other organs, to consist of round cells, large spindle-shaped or irregularly formed cells, epithelioid cells, giant cells (though these are not as numerous as in tubercles), fibroid interstitial tissue, and elastic tissue. Councilman[5] considers the essential process in the production of gumma in the lung to be a pneumonia with fibrinous exudation, accompanied by fibrous change in the alveolar walls, the whole subsequently undergoing caseation. There is a tendency to caseation and degeneration, beginning at the centre; young vessels spring from the periphery and grow toward the centre, and lead to absorption of the caseous nodule. If the absorption is complete a cicatrix is formed. It is also believed by some that the broken-down material may be discharged through a bronchus, and thus a cavity may result. Cavity formation is, however, very rare. That the anatomical diagnosis of pulmonary syphilis is very difficult and sometimes impossible has been, and still is, the opinion of the most experienced observers (Virchow, Ziegler,[6] and Ribbert[7]).

[1] Wien. med. Presse, 1880, Bd. xxi. No. 38.
[2] Berlin. klin. Wochenshr., 1875, No. 15.
[3] Dermatol. Zeitsch., 1893–1894.
[4] Ueber die Syph. Erkrankung der Lunge, München, 1901.
[5] Johns Hopkins Hospital Bulletin, 1891, vol. ii., No. 11.
[6] Spez. path. Anat., 1893.
[7] Lehr. b. d. Path. Histol., 1896.

Dürck and Hektoen[1] believe that a distinction can usually be made by the presence of newly formed bloodvessels in gumma. Sometimes, even in quite necrotic areas, vessels may be seen. Tubercle, on the other hand, as is well known, never shows new blood cells, and those old ones which may have been caught in the growth soon become occluded by pressure or thrombosis.

It is impossible to distinguish between tubercle and gumma in some instances, except by the finding of the tubercle bacillus. In a general way it may be said that in gumma fatty degeneration plays a very important part, while in tubercle the hyaline form is the more usual, and that giant cells are much rarer in gumma than in tubercle.

The pulmonary tissue immediately surrounding a gumma is frequently found to be consolidated, and from this infiltrated area fibrous bands may be seen extending to other parts of the lung.

2. Syphilitic Pneumonia of the Adult. It is pretty generally conceded that there is an infiltration of the lungs resulting from syphilitic infection, which alone, or in connection with nodules, may extend over a pulmonary lobe.

On section these infiltrated areas are of a dark slate or gray or grayish-red color, not granular.

Microscopically, as described by Aufrecht,[2] the alveoli were seen to be filled with cellular material, a few containing swollen alveolar epithelia exclusively; slightly more numerous were alveoli in which a small number of white blood cells were embedded in fibrinous masses, while the alveolar capillaries were distended with blood. But most of the alveoli appeared to be filled with small round cells interspersed with red blood corpuscles. A much larger quantity of the latter was present in the interalveolar and interlobular tissue. According to the same author, however, the only justification for considering this pneumonic inflammation as entitled to a special classification would be a considerable increase in the thickness in the walls of the smaller vessels.

3. Fibroid Induration. The proliferation of the connective-tissue elements may take the form of a thickening extending from the hilus around the bronchi and vessels, or of isolated masses of fibroid tissue in various parts of the lung, or of diffuse changes occupying the whole or the greater part of one lung.

This primary proliferation of the connective tissue in lung syphilis is considered by Pankritius[3] as being characteristic of lues, for, while in other diseases of the lungs which have a hyperplasia of the connective tissue, it is secondary to some primary condition, such as inhalation of dust, protracted croupous pneumonia, certain forms of tuberculosis, carcinoma, etc.

[1] Hektoen's Trans. of Dürck's General Path. Hist.
[2] Nothnagel's Cyclopedia of Practical Medicine.
[3] Hirsch, Jos. Inaugural Dissertation, München, 1901.

Microscopically this change is characterized by hyperplastic cellular proliferation of the interstitial, interalveolar, peribronchial, and perivascular tissues. The vessels show a thickening of the intima and the adventitia. The favorite seat of this form of the disease, as in the case of gummata, is said to be in the right middle lobe.

Virchow has pointed out that diffuse lung syphilis cannot be distinguished from the chronic pneumonia of stone-cutters and grinders.

Syphilitic Phthisis. *A Progressive Destructive Disease Due to Syphilis.* The possibility of such an entity as a result of syphilitic infection has not yet been satisfactorily proven. Osler says: "Personally, I have no knowledge of such an affection, either clinically or anatomically, and the cases which I have seen demonstrated do not seem to me to have characteristic distinction enough to separate them from ordinary tuberculous phthisis."

Until our knowledge of syphilis of the lung is much more exact than it is at present, we must agree with Fowler that before cases are put in this category they should fulfil the following conditions:

1. The cases must be complete—that is, the symptoms observed during life must be considered in connection with the lesions found on post-mortem examination.

2. The evidence of syphilitic infection must be undoubted.

3. Repeated examinations of the sputum must have been made, and the tubercle bacilli have been invariably absent, and the absence of tubercle from the lungs (as the cause of lesions) must be proved by post-mortem examination.

4. Syphilitic lesions about the nature of which there can be no doubt must be found in other organs. He gives several cases which seem to fulfil these conditions.

Of course, it is quite possible for a lobe or entire lung to be excavated as a result of more or less complete bronchial obstruction by a gumma, just as is occasionally seen in cases of aneurysm, but this is not due to the effect of the syphilitic virus upon the parenchyma.

It would hardly seem justifiable to classify separately certain changes observed in the bronchial glands and lymphatics in a case of syphilis of the lungs and other organs reported by Weber.[1]

Symptoms. Cough is usually the first symptom to attract attention, but has nothing characteristic. It depends more upon the laryngeal, tracheal, and bronchial lesions than upon those of the lung proper; at least, until these have become serious.

There is in many of these cases an associated ulceration of the larynx, trachea, or bronchi, which causes more or less constant irritation, resulting in cough.

Sputum. At first the sputum is that of an ordinary bronchitis, but later may become fetid from the bronchiectatic cavities formed by

[1] Pathological Society Transactions, vol. xvii. p 152.

the syphilitic changes in the lung. No tubercle bacilli are present in uncomplicated cases. There may, however, be elastic tissue depending upon the breaking down of the pulmonary parenchyma.

Hæmoptysis. This symptom, though not common, does occur, and is one of the most misleading, because it is so very usually a sign of tuberculosis (probably 5 out of 6 cases of pulmonary hemorrhage are due to tuberculosis). The amount of blood lost is not, as a rule, large, though a patient of Lancaux expectorated more than a litre in twenty-four hours.

Dyspnœa. This seems to be very usual. Of course, the frequently associated stenosis of the trachea and bronchi (pressure by gummata on cicatricial contraction) accounts for this symptom in many cases, but it sometimes seems out of proportion to the anatomical lesions. Mauriac explains its origin in these cases as due to flattening of the alveoli and degeneration of the alveolar epithelium. The shortness of breath is often paroxysmal and resembles at times bronchial asthma.

Pain. This is not considered an important symptom by the majority of observers.

General Symptoms. *Fever.* The temperature is of no value for diagnostic purposes, as it may be high or fever may be absent entirely. When present it may be of the hectic type, as in tuberculosis.

Night-sweats have been noted, but are not so common as in tuberculosis.

Emaciation, though not as frequent as in phthisis, may, in advanced cases, be most pronounced.

Physical Signs. There is, unfortunately, nothing distinctive. The signs produced by consolidation, whether from gummata or infiltration of syphilitic origin, differ in no way from those of tubercles or tuberculous infiltration.

Bronchiectatic cavities or excavations in the parenchyma produce the same signs, no matter from what cause.

Diagnosis. Some writers, especially the German, lay great stress upon the much greater frequency of syphilitic lesions in the middle lobe of the right lung. Grandidier, having observed this apparent preference in 27 out of 30 cases, considers it of the greatest importance in making a diagnosis. Schnitzler and Pankritius have made the same observation. On the other hand, Fowler[1] says: "A careful examination of undoubted specimens of pulmonary syphilis does not bear out the statement that lesions are generally limited to the middle part of the lung; they are so often found elsewhere that little importance attaches to the exact size in deciding the question of diagnosis."

A physical examination of the chest, taken with the symptoms,

[1] Loc. cit.

usually suggests tuberculosis, and this is the diagnosis made in the vast majority of cases. The absence of tubercle bacilli, after repeated examinations of the sputum, first suggests the possibility of syphilis. If a history of lues has not already been elicited, careful questioning may now bring it out, and a suspicion of the real cause of the trouble be aroused. The seat of the primary lesion may be found; the history of a cutaneous eruption, sore throat, or falling of the hair be given. Ulceration of the pharynx or larynx may be noted or probably luetic lesions of the bones, such as the sinking in of the bridge of the nose. Advanced arteriosclerosis in a young patient is also suggested. The signs and symptoms of stenosis of the trachea or main bronchi, where an aneurysm or growth can be excluded, may also strongly indicate syphilis as the probable cause.

PROGNOSIS. In early cases the prognosis is favorable. Unfortunately, a correct diagnosis is usually reached so late in these cases that the disease is many times too far advanced to admit of a cure. Whether antisyphilitic treatment will do much toward the removal of a gumma which is already in the advanced fibroid or cheesy state seems doubtful. The effect of treatment upon those chronic fibroid changes about the bronchi in the alveolar walls seems problematic in the extreme. The process may, however, be arrested. Bronchiectatic cavities are always serious, but when the obstruction is due to gummatous thickening, and not to fibroid contraction following submucous gummatous ulceration, it is probable that the condition may be cured; provided, of course, that it has not existed too long a time. If real cavity formation has occurred, the prognosis is unquestionably bad.

TREATMENT. Antisyphilitic treatment should be commenced without a moment's delay. The general trend of the present day is toward the use of mercury, as well as the iodides, in tertiary lesions, no matter where situated.

The administration of blue ointment by inunction and of potassium iodide by the mouth seems the best and most rapid way of combating this very serious disorder.

A great deal has been said about the occurrence of syphilis of the lung in conjunction with pulmonary tuberculosis. It may be said that it would seem the wisest plan, when such a condition is suspected, to institute the antisyphilitic treatment, with the hope of removing that infection at least. Cases have been reported which show that an antisyphilitic treatment may be carried out and the symptoms, such as roseola and ulceration of the pharynx, disappear without any ultimate bad results, so far as the tuberculous conditions are concerned.

The following report of a case of syphilitic disease of the lungs is of interest chiefly from a pathological point of view, as there were no symptoms which suggested the possible existence of the post-mortem findings:

William D., white, aged forty-four years; American; farmer. One uncle died of tuberculosis; otherwise the family history was good. He never drank whiskey and seldom beer; used tobacco moderately. At twenty-three years developed a sore upon the penis after exposure, the scar of which still remained. He received internal treatment at once and no secondaries developed. At thirty-nine years, sixteen years after the primary sore, he had a stroke, which paralyzed his right arm and the right side of his face. In about a year the paralysis was completely recovered from. Nine months ago he suffered from a swelling of the abdomen (probably ascites), for which he was treated and recovered.

For the past year he had been obliged to rise each night to urinate. For more than a month he had noticed that his feet were swollen, especially in the morning. Some months ago he ran a pitchfork into his ankle, making a small, punctured wound, which had never healed.

The present attack began, about two weeks before admission to the Garfield Hospital, with a cutting pain in the chest; a week later pain began in the abdomen. The feet and abdomen then became noticeably swollen, and there was dyspnœa on exertion. There was a cough with mucopurulent expectoration, which contained no tubercle bacilli.

The skin was pale, the eyelids puffy; there was general œdema. No thickening of the radials could be detected. The pulse tension was slightly plus. The heart was not enlarged; the second aortic sound accentuated; there were no murmurs. The respiratory sounds were harsh. The abdomen contained a fair amount of fluid. The thoracic and abdominal veins were distended. No enlargement of the spleen could be detected. The liver dulness seemed to be diminished, probably due to the floating up of the intestines by the ascitic fluid. The edge of the liver could not be felt.

Upon the sternum, between the junctions of the second and third ribs, was an irregularly shaped, flat swelling, which was not painful, giving to the examining finger a sense of hard elasticity. It was said to have followed a kick by a horse, and, as it was not recognized as a gumma, but little attention was given it.

The urine was scanty, averaging about 600 c.c. in the twenty-four hours. It was turbid, acid, specific gravity 1026, albumin two-thirds by bulk, no sugar; hyaline, fatty, granular, hyeloepithelial casts; a few leukocytes and blood cells. Ten and two-tenths grams of urea were excreted in twenty-four hours.

The urinary findings continued about the same through the course of the illness.

The blood showed 73 per cent. of reds, 8700 leukocytes, and 65 per cent. of hæmoglobin.

The œdema gradually became more pronounced, the ascites increased, and a left pleural effusion developed.

Treatment was of no avail, and the man died.

The clinical diagnosis was chronic parenchymatous nephritis, possibly of syphilitic origin.

I was present at the autopsy, which was performed by Dr. Butterfield. The notes read as follows:

Projecting from the sternum, between the second and third ribs, is an irregularly round nodule of firm consistence, not movable beneath the skin, about 3 or 4 cm. in diameter and 1.5 cm. in height. A pale, depressed scar, 1.5 cm. in diameter, on under surface of glans penis.

Inguinal glands enlarged and easily palpable. On the dorso-internal aspect of the left foot are two reddish areas covered with yellow crusts (scars from wounds mentioned in history).

Hypostasis in dependent portions. No evidence of cutaneous œdema. Parietal peritoneum thickened and opaque. Section of sternal nodule firm, yellow, surface smooth. It is firmly attached to sternum and adjacent ribs.

Pleural membranes thickened and opaque. Right cavity contains normal amount of fluid. Left cavity contains 45 to 60 c.c. of reddish serum.

Left Lung. Visceral pleura adherent at the apex anteriorly and posteriorly; also adherent between the lobes. On the anterior aspect of the upper lobe is a yellow nodule, 2.5 by 1.5 cm., adherent to the overlying costal cartilage. On section it appears to be encapsulated, extends down into the lung substance, is firm and is traversed by lines of pigment. The lung is crepitant throughout, slightly pale, and emphysematous along the anterior border and purplish-red at the base. Pigment deposits in septa; on section frothy serum exudes. Bronchi contain a mucopurulent material (cover-slip preparations show no tubercle bacilli). Weight, 505 grams.

Right Lung. Extensive adhesions at the apex, posterior portion of the upper lobe; also to diaphragmatic pleura. Pleura between the lobes adherent. On the inner aspect of the upper lobe is a yellow nodule, presenting the same general characteristics as that described in connection with the left lung. Lung crepitant throughout, pigmented pinkish. Bronchi contains mucopus. No tubercle bacilli demonstrable. Weight, 550 grams. No enlargement of the bronchial glands.

Pericardium. Parietal membrane thick, opaque. Cavity contains the normal amount of fluid. Heart apex is adherent at the base of the pericardial triangle. Epicardial membrane shows whitish opaque areas irregularly distributed over the heart. Myocardium thin, slightly flabby, and of a dark reddish-brown color. Aorta contains numerous atheromatous patches; on the aortic valve cusps are a few small vegetations. One leaflet of the mitral valve thickened and slightly contorted; the other slightly thickened. Other valves normal. Weight of heart, 241 grams.

Parietal peritoneum thick and opaque. Localized adhesions in some portions of visceral peritoneum. No excess of fluid. One large nodule in the omentum. No evidence of fat-necrosis.

Liver. Extensive adhesions to the diaphragm. Surface pale, marked by opaque, slightly depressed areas, a few mm. in diameter. Yellow encapsulated nodules in right lobe posteriorly. One extends into the liver substance for more than 3 cm. One very prominent nodule in the left lobe just at the fissure anteriorly. On section it is pale, firm, yellow, and showing a tendency to bulge in the centre of the nodule. Gall-bladder tense, with thick, greenish-black bile; not enlarged; no stones. Weight, 1668 grams.

Pancreas. Dense and enlarged; adherent to the spleen. Surface marked by numerous, whitish, opaque, miliary areas. Section firm; connective tissue between lobes increased and contains the same white opaque areas seen on surface of the viscus. Weight, 131 grams.

Kidneys. Right organ. Apex and adrenal body adherent to the liver; perirenal fat very abundant. Organ enlarged (12 x 6 x 4.3 cm.); surface smooth, pale, and mottled venæ stellatæ injected. Section showed no increased resistance on cutting. Cortex 9 mm. in width, pale, with yellowish areas. Pyramids congested, being of a dark purplish-red color.

Fat about the pelvis increased. Capsule strips readily. Weight, 293 grams.

Left Kidney. The same general characteristics as the right. Measurements, 13 x 6.5 x 4.5 cm. Weight, 294 grams.

Spleen enlarged, firm, but friable; surface smooth and capsule opaque. Section firm, deep red in color. Weight, 320 grams.

Stomach and intestines apparently normal. On the surface of the bladder there is a small nodule corresponding in consistence and color to those described in the liver, lungs, omentum, and upon the sternum.

Microscopic Findings. The nodule upon the sternum showed the usual picture of a gumma.

Sections of the liver showed marked cirrhosis, gummata, and amyloid degeneration. The kidneys were the seat of a diffuse nephritis and amyloid change. The pancreas showed pronounced cirrhosis of the interlobular variety and areas of fat-necrosis. No sections were cut from the nodule in the omentum, nor from that upon the bladder, but the macroscopic appearance of all the nodules was the same, and I have no doubt that they were all gummata.

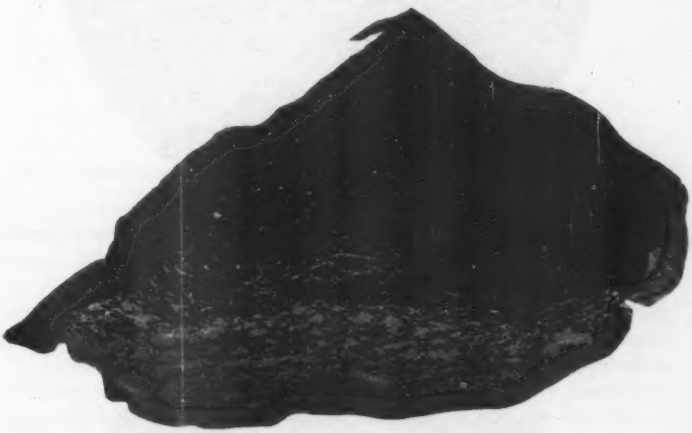
The Lungs. The two nodules described as being situated upon the anterior and inner aspects of the upper lobes of the right and left lungs, respectively, were found on section to consist of dense fibrous tissue, with necrotic centres (Fig. 1).

There was no distinct capsule on that portion of the growth toward the lung tissue. The alveoli seemed to fade so gradually into the

continuous fibrous growth that it was difficult to detect any distinct dividing line.

Numerous bloodvessels could be seen in the connective tissue (Fig. 2), and even a few in the necrotic area. This point is especially worthy of note, as it is considered by Dürck and Hektoen as characteristic of gumma in contradistinction to tubercle. The latter never show new bloodvessels. From the fibrous tissue of the nodule could be seen bands of connective tissue dipping down into the parenchyma of the lung, which in places showed a condition of catarrhal pneumonia (Fig. 3). On the outer aspect of the growth the fibrous tissue was continuous with the thickened pleura.

FIG. 1.



A section through part of a small gumma of the lung. × 10.

A number of sections were made from both lungs, which showed irregularly distributed areas of fibrosis and also small areas of pneumonia. These consolidated areas were too minute to be detected macroscopically, nor could they be felt. There was a thickening of the adventitious coats of the arteries, but none of the intima. As to whether or not there was any actual increase of fibrous tissue about the bronchi, I was unable to convince myself; it was certainly not universal.

The belief that the nodules in the lungs were gummata is based on the following: 1. History of a chancre, the scar of which remained. 2. The history of a paralytic stroke at thirty-eight years. 3. The presence of undoubted gummata of the liver and on the sternum. 4. The absence of tubercle bacilli from the sputum before death and at post-mortem. 5. The absence of bacilli from the sections of the nodules. 6. Extensive amyloid change in the liver, spleen, and kid-

FIG. 2.

Showing part of the new-growth, with granulation tissue rich in capillary bloodvessels at the margin. × 200.

FIG. 3.

3, Field beyond the fibrous area, showing a condition of catarrhal pneumonia and increased connective tissue in the area adjacent to the new-growth. × 125.

neys. 7. The absence of any tuberculous involvement of the apices or any other part of either lung. 8. The presence of numerous bloodvessels in the new-growth.

Although the presence of considerable black pigment would suggest a possible cause for the fibrosis in the lungs, it was noted that the fibroid areas apparently bore no constant relationship to the patches of pigment, and, therefore, it seems fair to conclude that the scattered areas of fibrosis and of consolidation were also of syphilitic origin.

I wish to acknowledge my indebtedness to Dr. James Carroll for his aid and advice in interpreting the microscopic findings.

SOME ASPECTS OF THE THEORIES OF IMMUNITY.

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On infection by bacteria it is the bacterial toxins which cause disease, the bacteria as such merely representing inert foreign matter. There are two distinct classes of bacterial toxins:

1. *Extracellular toxins* secreted by the bacteria during growth. Such toxins dissolve out into the surrounding medium, causing disease and death by toxæmia. Examples, diphtheria and tetanus.

2. *Intracellular or endotoxins*, which can hardly be regarded as secretions, but rather as a part of the bacterial body substance, being only liberated on the death and dissolution of the bacteria.

In the latter case the bacteria on gaining entrance to the circulation or body fluids may be destroyed at once, the amount of toxin thus liberated being too small to affect the organism, or the bacteria may be able to multiply; progressive increase and destruction going on *pari passu* until the number being destroyed is so great that the intracellular or endotoxins are liberated in sufficient quantity to cause manifestations of disease. Radziewski[1] was the first to point out this principle in a clear and convincing manner. An infection by such bacteria may be circumscribed and express itself as a local inflammatory process or it may be generalized in the circulation, in which case it is called septicæmia or bacteræmia. It will be convenient here to speak only of bacteræmia. Examples: Typhoid fever, staphylococcus and streptococcus infections.

The processes of immunization against these two types of infection are entirely different:

1. In toxæmia antitoxins are formed which neutralize the toxin, but do not affect the bacteria.

2. In bacteræmia no antitoxins are formed against the intracellular or endotoxins, but means are adopted by the organism for destroying the invading bacteria.

It is the second of these processes which it is proposed to discuss here.

The antibodies formed in bacteræmia are the agglutinins, the precipitins, and the lysins or destructive agents.

The role of the agglutinins in immunization is very obscure. It was at first supposed that agglutinated bacilli are more easily attacked by the lysins than normal bacilli. But this notion has not stood the test of experiments, and there is no good reason to assume that bacilli are rendered more susceptible to the action of the lysins by the fact of being agglutinated.

Again, it has been suggested that the agglutinins help to prevent growth of the bacilli, holding them in check until the lysins can act, but experiments do not bear out this theory either.

If immune serum, say typhoid, is heated to 56° C. for thirty minutes, its lytic action is destroyed, but its agglutinating power is not affected. If such heated serum in a test-tube is inoculated with a loopful of a typhoid culture, the bacilli will grow very readily, although they are agglutinated as they grow. They grow in fact quite as quickly in heated immune serum as in heated normal serum; so it is obvious that agglutination does not retard growth, *in vitro* at any rate.

What assistance, therefore, the agglutinins render we do not yet know, and the same may be said of the precipitins, but the part played by the lysins is more obvious.

As an introduction to the subject Ehrlich's well-known theory may be briefly sketched; a theory which endeavors to explain the mechanism of the lytic action of blood. It will not be necessary to describe the experiments of himself and others on the results of which the theory is based.

Ehrlich's Theory of the Lysins. *Normal Serum.* Normal serum is often strongly bacteriolytic for certain species of bacteria (typhoid, cholera, etc.).

The bacteriolytic action is due to two distinct principles acting in concert, the intermediate body (Zwischenkörper) and the complement. The intermediate body has an affinity for the bacillus, or rather for certain of the albumin molecules which go to make up the protoplasm of the bacillus, and enters into chemical combination with such molecules. The complement then enters into combination with the intermediate body and by virtue of its enzymatic nature can dissolve or destroy the albumin molecules of the bacillus to such an extent that the bacillus itself is destroyed.

It must always be borne in mind that the lysins are not directed against particular bacilli as such, but against certain albumins contained in the bacilli.

The complements exist in enormous numbers in the blood or fresh serum, but are of a very labile nature. On keeping serum for a few days the complements break up and disappear. Again, on heating fresh serum to 55° to 56° C. for thirty minutes the complements are destroyed. The intermediate bodies, on the other hand, are stable. They are not affected by keeping, even for months, or by warming until a temperature of about 65° C. is reached.

Serum from which the complements have been removed by keeping or warming to 55° to 56° C. is said to be *inactivated*. The intermediate bodies are still there and are able to attach themselves to the bacilli, but in the absence of the complements can exert no destructive influence upon them, and the bacilli grow freely in such serum.

The intermediate bodies are strictly specific—*i. e.*, an intermediate body which can combine with the albumins of the cholera bacillus is unable to combine with the albumins of the typhoid bacillus and *vice versa*. The complements, on the other hand, are not specific, although they may be multiple. Some may be better adapted for dealing with typhoid albumins, and others with cholera albumins, but there are probably many which can deal with various kinds of albumin indifferently. The question of the multiplicity or unity of complements, however, is still unsettled.

Immune Serum. On immunizing an animal to a bacillus, typhoid, for example, the complements are not increased in number. They remain numerically much the same as in normal blood, but the intermediate bodies for that particular bacillus multiply to an enormous extent, and are now known as *immune bodies*. It is not necessary to enter into the details of the mechanism by which Ehrlich accounts for the increase.

The immune animal, therefore, since its blood contains enormous quantities of immune bodies on account of the immunization, and enormous numbers of complements naturally, is able to dispose of enormous numbers of the particular bacillus to which it is immunized, although to other species of bacteria it is not, speaking generally, more resistant than it was in its normal condition.

This, in brief, is Ehrlich's theory, which, although leaving much yet unexplained, is the best concrete presentation of the subject that we possess so far, and is now very generally accepted.

Before proceeding to consider more in detail the lytic properties of immune serum, a digression may be made to point out that possibly the immunized animal is not more resistant to the action of endotoxins than it originally was in its normal condition.

A. Wolff[2] in recent articles has drawn attention to the analogy between bacterial endotoxins and the toxic principles of ordinary albumins. All inert albumins, when injected into an animal, are more or less toxic: red corpuscles, serum albumin, and egg albumen

very slightly; organ cells and especially spermatozoa more distinctly toxic.

Now, against the toxic principles of such albumins no immunity is conferred by repeated injections. On the contrary, the animal becomes more and more susceptible until it may succumb to a dose much smaller than would affect a normal animal. Moreover, the susceptibility is specific—*i. e.*, if an animal has become more susceptible to red cells, for example, it will resist an injection of spermatozoa as well as in its normal condition.

Agglutinins, precipitins, and lysins are formed against the albumins, but no antibodies which neutralize their toxic principles—*i. e.*, antitoxins. According to Wolff, the toxic principles of red corpuscles, spermatozoa, etc., are endotoxins, analogous to the endotoxins of bacteria. The endotoxins vary in the degree of their toxicity, but to all intents and purposes are similar in each instance, and no matter whether they are contained in the organ cells or in the bacterial cells, they are liberated on destruction of the cells and affect the animal organism in an analogous way. Since no anti-endotoxins are formed against them there is no increased resistance acquired on immunization. Some recent observations of my own seem to support these ideas of Wolff. Having had occasion to inject cancer extracts into rabbits, I found the animals stand the earlier injections, as a rule, very well, but soon lose flesh and often die after four or five infections, although the dose may be lowered rather than increased.

These rather sweeping statements of Wolff, however, cannot be accepted as proved. Although it may be taken for granted that no antiendotoxins are formed, it is usually supposed, and most experiments seem to bear this out, that some tolerance is acquired on repeated injections of organ or bacterial cells, just as tolerance to arsenic, nicotine, etc., may be acquired without the development of any antibodies.

It is generally accepted also that no antibodies can be formed against compounds simpler in structure than albumins, and this raises the question whether the endotoxins are actually of an albuminous nature or simpler substances against which one would not expect antibodies to be formed; a question which must be left undetermined for the present.

Whether the immunized animal is more resistant to the endotoxins than the normal is, therefore, still a moot point, but there is no doubt that a lethal dose *can* be reached in the immune animal by injections of large quantities of bacilli. In such a case the animal dies even though it may have destroyed every one of the bacilli injected (sterile death), so that at any rate the resistance to the endotoxins does not increase *pari passu* with the bactericidal power.

To return to the main argument we may take a rabbit, immunized to typhoid, and find that its serum can be inactivated at 56° C.,

mixed with living typhoid bacilli up to several times the lethal dose, and injected into the peritoneal cavity of a normal guinea-pig, or other animal, without injury to it. The immune bodies of the immune rabbit serum combine with the bacilli; the complements of the normal animal can now attach themselves to the immune bodies and, by dissolving out the albumins, destroy the bacilli. The immune serum can protect a normal animal against several lethal doses of typhoid bacilli. This may conveniently be called *passive* immunization as opposed to *active* immunization by inoculation with bacilli.

It is obvious that there is a minimum dose of immune serum which will protect. If there are not enough immune bodies injected to combine with all, or nearly all, of the bacilli, there may be more of the latter left alive than the normal animal can deal with on its own account and a fatal bacteræmia ensues.

So far all is plain sailing, but now difficulties begin to arise. The earlier experimenters, Löffler and Abel[3] in particular, observed that against typhoid bacilli not only is there a *minimum* dose of immune serum *below* which the serum will not protect, but there is also a *maximum* dose *above* which immune serum affords no protection. The accompanying table is given as an example. The injections were made into the peritoneal cavity:

TABLE I.

Immune serum and typhoid bacilli mixed and injected into guinea-pig. One loopful of bacilli is the minimum lethal dose (M.L.D.) per 100 grams of guinea-pig.

	Immune serum.	Bacilli per 100 grams of pig.	Result
1. Guinea-pig	0 control	4 M.L.D.	Dies
2. "	2 c.c.	4 "	"
3. "	1 "	4 "	"
4. "	0.5 "	4 "	Lives
5. "	0.25 "	4 "	"
6. "	0 1 "	4 "	Dies

The table only shows the principle, the amounts injected varying according to the strength of the immune serum and the virulence of the culture.

This was a phenomenon for which no explanation was forthcoming for some years after it was first observed.

In 1901 Neisser and Wechsberg[4] repeated these experiments, *in vitro*, and were able to demonstrate a similar occurrence by mixing immune serum with bacilli, adding a little fresh normal serum to supply complements (reactivation) and plating out. A table shows the results obtained by them better than explana-

tions. Each tube was made up to 2 c.c. by addition of physiological salt solution:

TABLE II.

	Typhoid immune serum inactivated at 56° C.	Emulsion of typhoid bacilli.	Fresh normal serum.	Colonies on agar plates made by taking five drops of mixture.	
				At once.	In five hours.
1 . . .	0	5 drops.	0.3 c.c.	Thousands	Thousands
2 . . .	1 c.c.	5 "	0	"	"
3 . . .	1 "	5 "	0.3 c.c.	"	"
4 . . .	0.5 "	5 "	0.3 "	"	Hundreds
5 . . .	0.25 "	5 "	0.3 "	"	0
6 . . .	0.1 "	5 "	0.3 "	"	Hundreds
7 . . .	0.05 "	5 "	0.3 "	"	Thousands

Neither the normal serum (1) alone at the dilution given, nor the immune serum (2) alone would kill any typhoid bacilli, but properly combined (5) they killed immense numbers. An excess of immune serum (3), however, is just as ineffective as too little (7).

These observations both *in vivo* and *in vitro* have been repeatedly confirmed and no one now doubts their correctness.

Neisser and Wechsberg's explanation is that the complements combine directly with the immune bodies, and this combination then attaches itself to the bacillus. If, then, the immune bodies are in excess of the complements, some of the immune bodies will be unprovided with complement, and these may attach themselves to some of the bacilli to the exclusion of the complement—immune body combinations. Such bacilli would escape unharmed, and, multiplying, set up a fatal bacteræmia.

This is called Neisser and Wechsberg's phenomenon or diversion of complements (Komplement-Ablenkung). Not only can complement diversion occur as explained, but it is also a fact that fresh immune serum alone *in vitro* will often not kill its specific bacillus. The following table gives an example from among my own experiments:

TABLE III.

Fresh rabbit serum eighteen hours old; 1 c.c.	Bacilli.	Number of bacilli per cubic millimetre estimated from colonies on agar plates.		
		At once.	In five hours.	
1. Normal.	Typhoid.	1000	0	The bacilli have been killed.
2. Serum of same rabbit after immunization to typhoid.	Typhoid.	1000	2000	The bacilli have multiplied.

This is probably another instance of complement diversion.

We find, then, that immune serum may not kill its own specific bacilli *in vitro*, and that an excess of immune serum may not protect a normal animal. In the latter case there must be complement diversion *in vivo*, yet if we take an immune animal and inject large quantities of the bacilli of immunization the bacilli are always destroyed; so it does not seem that there can be any complement diversion in such cases.

The destruction may even be on such an extensive scale that the immune animal is actually killed by the endotoxins liberated. Why should there be complement diversion in the case of the passively immunized, and apparently none in the case of the actively immunized animal?

Consideration of this question will entail a rather lengthy discussion, throughout which the main point must be kept in view or the thread of the argument will be lost.

Is it possible that in the immune animal itself the complements are locked up in some way so that they cannot combine with the immune bodies until after these have become attached to the bacilli? Supposing, for instance, as Metchnikoff[5] holds, that in the ordinary way the complements are contained in the leukocytes and not liberated at all, the bacilli becoming loaded with immune bodies and then taken up by the leukocytes, which digest them by virtue of the complements which they contain.

Metchnikoff's theory can now be taken up to see if it will help to solve the difficulty.

Metchnikoff's original idea, derived from observations on Daphnia and other lower organisms, was that it is the polynuclear leukocytes alone which destroy bacteria by means of phagocytosis. This is the purely cellular theory as opposed to the purely humoral theory that the bacteriolytic agents only exist free in the blood plasma or serum. When it was proved beyond all doubt that the immune bodies and complements do exist free in the serum, of vertebrate animals at any rate, Metchnikoff had to modify his views. He now admits that the immune bodies are secreted and dissolved out into the blood plasma, probably by the leukocytes, though possibly by other cells also. But the complements (his cytase) are formed by and retained within the leukocytes, and are only liberated on injury to or death of the latter. The complements do not normally exist free in the blood or body fluids.

The immune bodies, therefore, are analogous to the extracellular toxins and the complements to the endotoxins of the bacteria.

Metchnikoff bases this theory mainly on certain experiments made under his direction by Gengou,[8] who drew blood into paraffined tubes, centrifugalized quickly before the blood coagulated, and drew off the plasma. The plasma so obtained was much less bactericidal than serum obtained in the ordinary way. If the

blood is allowed to clot and the serum drawn off, there is destruction of leukocytes and the complements are liberated. If the plasma is taken by Gengou's method there is no or very slight destruction of leukocytes; so the plasma is not at all or very slightly bactericidal. Proof 1.

Again, Gengou found that leukocytic extracts, obtained by injecting starch suspensions into the pleural cavities of dogs or rabbits and withdrawing the sterile exudates, were strongly bactericidal after keeping for twenty-four hours at 37° C. Many of the leukocytes have perished and liberated the complements. Proof 2.

Now, experiments with immune animals are usually made by injections into the peritoneal cavity. In such cases, says Metchnikoff, the injection of comparatively large amounts of fluid causes *phagolysis*, or destruction of leukocytes, so that complements are set free and the bacilli killed, extracellularly at first to some extent, but in a short time fresh leukocytes arrive which take up the bacilli still remaining, now loaded with immune bodies, and complete the digestion by means of the complements (cytase) contained in them.

Metchnikoff, who, be it said at once, is not arguing all this as an explanation of the difficulties of explaining complement diversion, therefore takes us almost back to the purely humoral theory, and has to admit that destruction of bacteria may occur extracellularly *in vivo*, as well as in the interior of the leukocytes. Still, he lays great stress on the importance of phagocytosis, and we may suppose that after the primary extracellular destruction of bacteria, during which there may be some diversion of complements in both the actively and passively immunized animal, the phagocytes in the former case arrive more promptly and are more active than the phagocytes of the passively immunized animals. That the phagocytes in the *blood* of an immune animal are more effective than those of a normal animal will be shown later. Consequently the bacilli which have escaped extracellular destruction on account of complement diversion are taken up by the leukocytes later on: *in toto* by those of the actively and only partially by those of the passively immunized animal.

For this reason, although there may be an excess of immune bodies and some complement diversion in either case, the actively immunized animal lives and the passively immunized one dies.

In the animal or patient, self-immunized on account of natural infection, there would be no destruction of leukocytes, and, therefore, no chance for diversion of complements, since these would not be liberated.

This, however, is not a very satisfactory explanation and becomes of doubtful significance when we find it quite possible that Metchnikoff has based his conclusions upon erroneous premises.

1. Experiments with Plasma. Although a great number of observers agree with Gengou that plasma is less bactericidal than

serum, there are many who deny this. Vedder,[7] to mention one author, followed Gengou's methods very closely, and came to the conclusion that the plasma so obtained was just as bactericidal as the serum, no more and no less. It is, therefore, by no means certain that the complement content of serum is due to destruction of leukocytes during clotting.

2. Leukocytic Extracts. Gengou's method of obtaining leukocytic extracts was as follows: He drew the leukocytic exudate from the pleural cavity and centrifugalized. The fluid above was then drawn off, the leukocytes washed twice, allowed to stand for twenty-four hours at 37° C., and then again centrifugalized. The clear fluid drawn off from above the washed leukocytes was bactericidal. Ergo, the complement had been liberated from the leukocytes as they disintegrated. The accuracy of this observation has also been challenged. Petrie,[8] for example, working with an improved technique, washed the leukocytes four times, and then ground them to pieces at the temperature of liquid air, —180° C. The extracts so obtained were not in the least bactericidal, nor would they reactivate inactivated immune serum, as complement-containing serum will do.

Therefore, he decides that the leukocytes do not contain complement, a conclusion directly opposed to that of Gengou. Petrie points out three sources of error in Gengou's methods:

(*a*) The leukocytes were only washed twice. If Petrie only washed twice, his extracts were somewhat bactericidal. This was because the complement-containing fluid portion of the exudate had not all been washed out. The fact that the partially washed extracts were bactericidal also shows that the low temperature of —180° C. is not injurious to complements.

(*b*) Complements are very labile, and keeping at 37° C. for twenty-four hours would weaken them considerably.

(*c*) When cell suspensions are kept at 37° C. for twenty-four hours they will undergo some self-digestion (autolysis). Now, Conradi[9] has shown that fresh organ extracts, liver, spleen, etc., are very slightly, if at all, bactericidal, but after a short period of self-digestion they become markedly so, not on account of increase of labile complement, but of stable basic products of the autolysis.

Petrie, therefore, decides that if the bacteriolytic action of Gengou's extracts was due to complement, it was because the fluid of the exudate had not been thoroughly washed out. But in all probability it was not due to complement at all, but to basic products of autolysis.

Petrie's contribution is undoubtedly of great importance and shows that at the best we must accept Gengou's conclusions with considerable reserve. It cannot be taken as proved that leukocytes contain complement which is liberated on their disintegration.

Careful experiments by several recent observers, notably A. Wolff,[10] show, that on injection of bacilli into the peritoneal cavity of immune animals, the bacilli are all destroyed extracellularly in a very short time, before the arrival of the leukocytes. These come in later, nevertheless, but there is *nothing left for them to do* except to carry off the remains. There seems to be little doubt that this is the case.

Again, the later experimenters have been quite unable to demonstrate that there is any phagolysis on injection of fluids or suspensions of bacteria into the peritoneal cavity; so it is very doubtful if Metchnikoff is justified in asserting that phagolysis occurs.

Phagocytosis, therefore, according to A. Wolff and others, plays only a secondary part in bacteriolytic phenomena, and is altogether absent in the peritoneal cavity of the immunized animal.

Seeing to what an extent various authors differ in their views, we must hesitate to accept one theory to the exclusion of others, although so far as the peritoneal cavity is concerned it seems most probable that the role of phagocytosis is comparatively unimportant. The latest and most careful observations tend to support this view.

Although we cannot attempt in the present state of our knowledge to explain by experimental evidence why diversion of complements occurs *in vitro* and in passively immunized animals, while in actively immunized animals apparently no such diversion takes place, yet we must remember that once blood is shed the condition for bacteriolytic processes are artificial, and it is quite possible that in the serum the affinities of the immune body and complement are increased so that they will combine more readily than in the immune animal itself. If this is so it would account for the phenomena observed. But it is pure hypothesis. There is no experimental proof of it.

In any case the peritoneal cavity is not an ideal spot to gain information about infection and immunity. The fight between the organism and the bacteria goes on for the most part in the circulation if the infection is general. The blood, therefore, is likely to afford a better insight into the processes involved, and within the last two or three years a new school has arisen—the opsonic school of Wright, as it may be called from its founder, which bases its theories mainly on observations of what appears to go on in the blood.

In 1902 Leishman[11] published a method by which the phagocytic action of leukocytes may be observed *extra vitam*. He takes a drop or two of blood, mixes it with an equal quantity of an emulsion of bacilli on a slide, and puts a cover-glass on it. The specimen is allowed to stand for thirty minutes in the incubator and then a smear is made and stained. The polynuclear leukocytes are seen to contain bacilli; those of normal blood a few; those of immune blood a large number. We may suppose, for instance, that the

average number of bacilli in each leukocyte of normal blood is four or five. It is found that in a parallel experiment made with immune blood the number may average fifteen to twenty in each leukocyte.

Wright[12] and Bulloch[13] have made extensive observations on this phenomenon and have come to the conclusion that there is some principle, opsonine (feast preparer), in immune blood which enables the leukocytes to take up and digest the bacilli more readily than normally. This principle resides in the serum, for if the leukocytes of the immune blood are washed free of serum with salt solution they are unable to take up any quantity of bacilli; not more can be taken up by the leukocytes of normal blood.

The opsonine, so far as has been determined, does not destroy the bacilli extracellularly; so its action is not analogous to that of the complements and immune bodies, but, like the latter and the agglutinins, it can attach itself to the bacilli.

For instance, if on the one hand bacilli are treated with immune serum, washed free of serum, and suspended in salt solution, while on the other hand leukocytes are washed and suspended in salt solution, the two suspensions can be mixed, and the leukocytes will take up the bacilli now loaded with opsonine just as freely as in immune blood. Wright does not enter into the question of the relations between opsonine and the lysins. This is left undetermined for the present, but we may consider it probable that whatever may go on in the peritoneal cavity, in the blood phagocytosis must play a leading part, and the latterly somewhat discredited phagocytic theory of Metchnikoff may obtain a renewed lease of life from a new point of view.

Therapeutics in Bacteræmia. While the principles involved in the foregoing discussions are of great theoretical interest, the ultimate object of research work in this field is to find some therapeutic agent for bacteræmic infections.

The success of the antitoxin treatment of diphtheria led to the hope that all bacterial diseases might be beneficially treated with antiserums. With this idea in view experiments were made on a large scale with antityphoid, antistreptococcus, antipneumococcus, and other antiserums, but with little or no success.

The reasons for failure are not far to seek. Such serums contain no antiendotoxins, so would not neutralize the endotoxins, although they would assist bacteriolysis if injected into a normal animal together with their specific bacterium. If used on a patient, however, the serum would be taken from stock in hand and, therefore, not fresh, so that the complements would have disappeared, leaving only the immune bodies. But the patient's blood is probably already loaded with immune bodies, so that the injection of a further quantity could do no good and might do harm if there were any tendency to diversion of complement, owing to intro-

duction of artificial conditions. It does not seem likely that any such antiserums will ever be of use.

Suggestions have frequently been made that to inject the fresh complement containing serum of an animal might lead to good results. In all probability, a typhoid patient, or one suffering from an analogous infection, has an excess of immune bodies and a deficiency of complements in his blood, and by supplying more of the latter the bacilli could be destroyed more rapidly. It is by no means certain that this would be the case, but, granting that it is so, an element of danger may arise. On sudden destruction of large numbers of bacilli, such as might occur on injecting a large number of complements, endotoxins may be liberated to such an extent as to cause an aggravation rather than an abatement of the fever or other symptoms, or even lead to the death of the patient.

In the present state of our knowledge the dosage of complements would be altogether empirical. The complement content of serums varies so much that it would be impossible to estimate the amount of complements injected at any one time, nor could one judge of the quantity required to do good without introducing the element of danger.

VACCINES. Wright claims that the opsonic power of the blood can be increased by injections of killed cultures (vaccines), and has treated several cases of carbuncle, resulting from staphylococcus infection, with apparently beneficial results.

The cultures of staphylococcus are killed at a temperature of 60° C. and the sterile suspension injected into the patient. The serum of such patients may not, and usually does not, kill one single staphylococcus *in vitro*, but examination of the blood by Leishman's method before and after injection shows that the power of the leukocytes to take up the cocci is very greatly increased. Coincidently with this increased opsonic power of the blood the patient improves in health. The same has been observed with tuberculous patients after injection of tuberculin.*

Wright[14] has also used this method as a prophylactic against typhoid fever and has succeeded in standardizing the dosage of vaccine with considerable accuracy. As a result of two or three injections into a healthy subject the opsonic power of the blood for typhoid bacilli is notably augmented and it is inferred that this indicates an increased resistance to infection.

During the Boer War Wright's vaccines were largely used among the British soldiers, but statistics, more or less carefully kept, did not bear out the claims of the originator for the treatment, and it has been abandoned by the war-office authorities.

Wright intimates that it was the statistics and not the treatment which were at fault. However this may be, it seems likely that

* Lancet, October 10, 1904.

future prophylactic and curative measures against bacteræmia will be along the lines of vaccines rather than antiserums, yet in all probability a considerable period must elapse before the bacteriologist will be of much assistance to the clinician in his treatment of such infections.

REFERENCES.

1. Radziewski. Zeit. Hygiene, 1901, Bd. xxxvii. p. 1.
2. Wolff, A. Berl. klin. Woch., 1904, Nos. 42, 43, 44.
3. Löffler und Abel Centralblatt f. Bacteriologie, 1896, Bd. xix. p. 51.
4. Neisser und Wechsberg. Münch. med. Woch., 1901, No. 18.
5. Metchnikoff. Études sur l'immunité, 1901.
6. Gengou. Annales Pasteur, 1901, tome xv.
7. Vedder. Journal of Medical Research, 1903, vol. iv. p. 475.
8. Petrie. Journal of Pathology and Bacteriology, 1903, vol. ix. p. 130.
9. Conradi. Hofmeister's Beiträge, 1902, Bd. i. p. 193.
10. Wolff, A. Berl. klin. Woch., 1903.
11. Leishman. British Medical Journal, January 11, 1902.
12. Wright, A. E. Ibid., September 10, 1904; Lancet, 1902, 1903, and 1904.
13. Bulloch. Ibid., September 10, 1904.
14. Wright, A. E. Antityphoid Inoculation, 1904.

RECURRENT OCULOMOTOR PALSY, WITH A REPORT OF A CASE.[1]

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RECURRENT oculomotor palsy is a rare affection, although cases have been reported in this country by de Schweinitz,[2] Leszynsky,[3] Sachs,[4] Weber,[5] and Onuf.[6] The reports in most of these cases have been very brief, and it is doubtful whether, according to the distinctions of Möbius, most of these cases should be regarded as typical. The following case should be classed in the symptom group.

On October 31, 1904, C. C. N., a physician, aged thirty-one years, consulted one of us (Dr. Posey) on account of double vision and drooping of the right lid. His eyes had been refracted five years previously, while he was in attendance at the medical school, because of recurrent attacks of pain in and over the right eye, but nothing abnormal in the eye grounds or in the extra-

[1] Read before the Ophthalmological Section of the College of Physicians of Philadelphia, December, 1904.

[2] Boston Medical and Surgical Journal, 1895. [3] Medical Record, May 25, 1901, p. 812.

[4] Journal of Nervous and Mental Disease, 1901, p. 462.

[5] Ibid. [6] Ibid.

ocular muscles had been noted at that time. The patient still wore the glasses which were then given him for the correction of a moderate amount of hypermetropic astigmatism, and which for a time had relieved his symptoms perfectly. When the eyes were examined for the purpose of ascertaining the cause of his present difficulty, an almost complete ptosis of his right upper eyelid was at once manifest, while further study elicited that the inferior oblique and the internal rectus muscle of the same eye were paretic. The upper lid and the extraocular muscles of the left eye were unaffected, and the irides and ciliary muscles acted normally in both eyes. Corrected vision was also normal in both. An examination of the nose and its accessory sinuses by Dr. Freeman revealed nothing abnormal.

The diagnosis pointed to a partial palsy of the external division of the right oculomotor nerve; upon questioning the patient, however, in order to ascertain the cause of the palsy, a series of facts was developed which at once surrounded the case with unusual interest and indicated that the character of the affection was quite out of the common. The family history of the patient was good, his father being still alive at the age of seventy-five years; his mother having died of splenic anæmia when forty-nine years of age. Four brothers and one sister are living and well, one brother having died of acute pancreatitis at thirty-six years of age. There is no history of nervous or neurotic disease in the family, nor, as far as the patient could ascertain, had any of the members been the subject of an ocular palsy. His sister is rheumatic and suffers from migrainous attacks, but all his brothers are free from them.

In early childhood the patient had measles and had repeated attacks of lumbago, which have persisted with a moderate degree of severity until the present time. When he was about fourteen years of age he began to suffer from spells of dimness of vision, associated with flashes of light upon the side, and followed by headache and nausea. These seizures apparently were typical attacks of migraine. They occurred upon both sides of the head and were brought on by gastric disturbances, overuse of the eyes, and general fatigue. At the end of two years the character of the attacks changed as the spectra disappeared, though the pain in the head and the gastric symptoms still persisted. While in attendance at the medical school, the pain in the head was almost constantly present, especially over the right eye, which led him, as mentioned above, to seek relief from glasses. His health is otherwise good and gonorrhœa and syphilis are denied.

In January of this year, after continuous professional work of more than usual severity, following an attack of pain in the right eye which had persisted for a few days, he saw double for the first time. He is unable to say whether the attacks of migraine had been more aggravated just prior to the attack of diplopia than usual, but he is confident of the preceding pain in the eye without

the migrainous symptoms. At the time of the attack the double vision was more marked when he looked to the left and above, but there was no ptosis. This diplopia persisted several weeks and

FIG. 1.

Left eye fixing an object above. Right eye is deviated downward, and the lid is drooping.

FIG. 2.

Left eye fixing an object far to the left and slightly above the horizontal visual plane. Right eye is deviated down and in, as a consequence of a paretic condition of the right inferior oblique and internal rectus muscles.

then gradually passed away, leaving no apparent ocular disturbance, his eyes giving him no further trouble until July, when a return of the double images was again remarked. The double images were of much the same character as in the preceding attack, but there was the additional symptom of the drooping of the upper lid of the right eye. At first the ptosis was intermittent, but in the course of a few weeks it became permanent and almost complete, until November 1st, when the lid raised for several days, but dropped again, however, and the eye remained closed until November 24th, when the lid again raised and resumed its natural appearance and so continued one week, when it began to partially droop, in which condition it still remains.

On account of the unusual course of the palsy, the attack of diplopia six months previous to the onset of the present palsy being also occasioned in all probability, as judged by the position of the double images, by an involvement of the right aculomotor nerve, it was thought best to ascertain whether there existed any further involvement of the nervous system. The patient was accordingly examined by one of us (Dr. Spiller), who found that he closed his eyelids well, drew up the corners of the mouth fairly well together, but not very well separately. The tongue was protruded straight, was moved freely, and showed no fibrillary tremors. The masseter contracted well on each side. Sensation for touch and pain was normal in the two sides of the face.

He had no weakness of the limbs. The patellar reflexes were prompt but not excessive, and the right was a little prompter than the left. The Achilles jerks were prompt but not excessive, and he did not have ankle clonus. Sensation for touch and pain was normal in the hands and feet. The gait and station were normal with eyes open or closed. He had possibly an area of slight hypalgesia on each side of the thorax about the nipple line; this, however, was not positive, and tactile sensation here was normal.

The patient when questioned gave further information regarding the attacks that he had had when he was about fifteen years of age and later. In these he would become completely blind, and would not recognize an object across the room, although he could see light faintly. These attacks lasted about half an hour, and were followed by severe headache. During the blindness he saw flashes of light. The blindness disappeared before the headache developed. He had not had any of these attacks during the past ten years.

Möbius gives a description of recurrent oculomotor palsy somewhat different from that of most writers. He presents a brief summary of all the cases reported since 1895, but of these only 6 are regarded as typical. They are the cases of Karplus (3), de Schweinitz, Ballet, and Paderstein. The symptoms begin in youth, at least before the twenty-fifth year. There is no heredity and the patients do not usually come from families in which migraine

occurs, although the occurrence of migraine in the family is not sufficient to prevent a diagnosis. The attacks always begin with headache, vomiting, or nausea, and, as a rule, the headache ceases when the paralysis develops, but it may last a few days after the paralysis has begun. There may be vertigo, increased flow of saliva, and vasomotor symptoms. Flimmer scotoma does not occur. The paralysis never develops without migraine, but there may be attacks of migraine without paralysis, and the headache is always on the same side. The migraine may last hours or weeks; the paralysis weeks or months; indeed, the headache is likely to last longer than in the simple migraine. The frequency of the attacks varies, there may be several attacks in one year, or several years may intervene between the attacks, but there is a type for every case. The periodicity is like that of epilepsy, migraine, and periodic insanity. The oculomotor paralysis is unilateral and complete, never confined to the external ocular muscles, or to the internal. Between the attacks some weakness of the oculomotor supply persists, and in course of time the residual paralysis increases. The first branch of the trigeminus has been found implicated.

Möbius combats Charcot's opinion that the periodic oculomotor palsy is a form of migraine (migraine ophtalmoplégique), and he believes the migraine is only symptomatic, as it may be in epilepsy or paretic dementia. Migraine may cause oculomotor palsy, but in periodic oculomotor palsy the lesion causes the palsy and the migraine is an aura. How migraine may cause the palsy is unknown. In most cases of periodic oculomotor palsy the paralysis occurs in the first attack.

Möbius[1] assumes that the lesion is at the base of the brain. He mentions that de Schweinitz has reported recurrent abducens palsy.

Oppenheim[2] gives a more liberal interpretation to the symptom-complex. According to him the periods may be irregular, all the oculomotor supply need not be implicated, and the paralysis may be confined to one muscle, as the levator palpebræ superioris in Knapp's case. The second branch of the trigeminus may be affected. In the intervals between the attacks there may be no paralysis. In the cases in which a necropsy has been obtained (Ziehen, Weiss, Thomsen-Richter, Karplus) basal hemorrhagic pachymeningitis was found in one case, a tubercle, a fibrochondroma, and a neurofibroma in the others. Oppenheim thinks that the periodic oculomotor palsy is related to migraine, as Charcot believed, and dependent upon vasomotor disturbance. Spasm of the vessels obstructs the blood supply and causes the paralysis, or overfilling of the vessels compresses the nerve. Attacks may occur without permanently injuring the nerve, but finally degeneration

[1] Deutsch. Zeitschrift f. Nervenheilkunde, 1900, Bd. xvii. p. 294.
[2] Lehrbuch der Nervenkrankheiten, third edition.

and inflammatory changes occur in the nerve and the paralysis persists. Such vascular disturbances may cause exudative processes or tumor. The symptoms may be progressive, or may be arrested, or may disappear.

Some very important distinctions will be noticed between the descriptions of Möbius and Oppenheim. A lesion may readily cause a paralysis of only certain branches of the oculomotor nerve, even though it be a tumor at the base, and a partial palsy should not be sufficient reason for exclusion of a case. The limitation to an age below twenty-five years is certainly arbitrary.

The case that we have studied is especially interesting in regard to the migrainous attacks. When about fifteen years old and later the patient had ophthalmic migraine—*i. e.*, attacks of blindness accompanied by flashes of light—but these had ceased about ten years before he had the oculomotor palsy. The case is evidently one of recurrent oculomotor palsy, and while it does not strictly correspond to Möbius' narrow limitations in the partial palsy, the age of the patient and the preceding attacks of migraine, it does illustrate the symptom-complex as given by Oppenheim. Why, we may ask, should the palsy not be partial even though the cause is an organic one, why should the symptoms not appear at the age of thirty as well as of twenty-five; why should migraine not precede the palsy even for many years? The condition is a symptom-complex not a disease, and the pathology has been determined in only four cases. Indeed, it is questionable whether Ziehen's case with necropsy should be regarded as one of recurrent oculomotor palsy. Is there good reason for excluding such a case as the one reported by Leszynsky?[1]

A woman, aged twenty-nine years, began when six years old to have attacks of headache confined to the right temporal and supra-orbital regions, and invariably accompanied by vomiting. The attacks occurred every five or six weeks. At her twelfth year the headache was associated with ptosis of the right eye, from which she recovered in two weeks, the migraine continuing to occur as before. The second attack of oculomotor palsy occurred in her nineteenth year, with the same kind of pain and vomiting. She had partial ptosis, diplopia, and inability to look upward with the right eye. She improved in three weeks, but the eye did not move upward as well as before for a few months, and then motility was completely regained. The third attack occurred in her twenty-second year, and was characterized by almost complete ptosis, outward deviation of the eyeball, and diplopia. She recovered in about a year. She had five attacks.

It is well to define a symptom-complex sharply, but in the present state of our knowledge regarding recurrent oculomotor palsy,

[1] Journal of Nervous and Mental Disease, 1901, p. 462.

when the ignorance of the pathology is so great, the description may be made too narrow.

One must always remember that a recurrent oculomotor palsy may be the first sign of a general organic disease of the nervous system, like tabes or multiple sclerosis, but in the case we have reported no sign of such disease could be found.

MYASTHENIA GRAVIS, WITH PARALYSIS CONFINED TO THE OCULAR MUSCLES.[1]

By William G. Spiller, M.D.,
of Philadelphia,
and
Ernest U. Buckman, M.D.,
of Wilkesbarre.

Myasthenia gravis may present symptoms of implication of the ocular muscles in association with other signs of the disease, but a limitation of the weakness to the ocular muscles is exceedingly uncommon, and cases of this character are not always easy to diagnosticate. W. Sterling[2] has recently reported a case in which the paresis was observed only in a drooping of the upper lids, the ptosis varying in intensity from time to time. The myasthenic reaction was obtained in the biceps, deltoid, and sternocleidomastoid muscles. This was the only case of this character Sterling knew of, but one of us (Spiller), in association with Dr. William Campbell Posey, has observed a similar case, in which bilateral ptosis of varying intensity was the only sign of the disease, except that one other ocular muscle was slightly affected. Dr. Posey will probably report this case. Sterling refers to the cases of Camuset, Karplus, Kunn, and Wilbrand and Sänger, in which the weakness was only in the ocular muscles, although in the first two cases other symptoms of the disease developed later. The myasthenic reaction to the electric current does not seem to have been obtained in any of these four cases.

A case reported by Oppenheim is as follows:

A man, aged forty-seven years, not syphilitic and not alcoholic, in February, 1904, noticed suddenly diplopia. He had some headache during a few days. He was strong, and only the ocular muscles were affected. Both internal recti muscles were paralyzed, and within a few days ptosis, especially on the right side, developed. The intensity of the ptosis varied greatly; at times it was only slightly indicated,

[1] Read before the Philadelphia Neurological Society, January 24, 1905.

[2] Monatsschrift f. Psychiatrie und Neurologie, vol. xvi. p. 183. (Ergänzungsheft.)

at other times the pupils were almost covered by the upper lids. The levator palpebræ superioris, therefore, appeared to be easily exhausted. If the man were made to look upward, the upper lids began to fall within a few seconds. The action of other muscles (presumably ocular) caused exhaustion of the levator. Occasionally the ptosis was more pronounced on the left side. He had neither diplopia nor ptosis in the morning during an hour after awakening. Myasthenic reaction was observed in the left deltoid. This muscle was not fully exhausted by the electric current, but the contractions became weaker with every irritation. There were no other symptoms of involvement of the nervous system at this time. The right large toe was unusually large. Later bulbar symptoms developed, and the disease terminated fatally.

Oppenheim remarks that these ocular forms of myasthenia gravis have not received sufficient attention, and that probably they are more common than is supposed. The diagnosis of cerebral syphilis or tabes is likely to be made in such cases. Tabetic ptosis may be a hypotonia of the levator palpebræ superioris, and may disappear for a time, and variation in intensity of the ocular palsy is recognized in tabes. The signs of tabes at first may be in the ocular muscles and may be confined to these for a long time. The internal muscles of the eyes are not affected in myasthenia, but these muscles may also escape in tabes. The myasthenic electric reaction is a positive means of diagnosis.

Oppenheim[1] points out that congenital defects have been observed in myasthenia, such as the enlargement of the toe he observed in his case.

The case of myasthenia gravis we report is remarkable, in that the weakness was observed only in the ocular muscles. The rapid exhaustion of the levator palpebræ superioris, first of one side and then of the other, especially when both eyes were uncovered at the same time; the recovery after rest, the variation in the paralysis of other external ocular muscles of the oculomotor distribution, the integrity of the inner muscles of the eyeballs, a response of the sternocleidomastoid muscle to the faradic current, suggesting the myasthenic reaction, and the absence of all other signs of implication of the nervous system, make the case probably one of myasthenia gravis of the ocular type.

Notes by Dr. Buckman, January 8 *and* 13, 1905. C. F. L., aged thirty-three years, a groceryman, muscular, consulted me April 26, 1904, on account of dizziness and blurring of sight, which had lasted about one week. His vision was, and now is, about normal. He complained that everything looked crooked with both eyes, but perfectly normal with either alone. The slight error of refraction

[1] Deutsch. med. Wochenschrift, July 14, 1905, p. 1053; Berliner klin. Wochenschrift, February 6, 1905, p. 164.

was corrected under atropia, and he was ordered for the right eye +.25 ⊂ + .25 c. ax. 90°; for the left eye, + .50 ⊂ + .25 c. ax. 90°, combined with prism 2° base up and ½° base out. This correction lasted but a short time, and the muscular deviation gradually increased. He could use either eye alone, but could not use both eyes together, and has not been able to do so since that time.

On November 14th there was almost complete paralysis of the inferior rectus muscle of the right eye. The condition improved under large doses of iodide and daily applications of electricity. In a short time from the last date ptosis of the left eye developed, with occasional ptosis of the right. He took mercury to ptyalism and is now taking large doses of strychnine.

Sometimes one muscle is paralyzed and sometimes another. The sixth nerve has never been affected. He has not had optic neuritis, nausea, vomiting, headache, nor vertigo during the past six months. He has not had syphilis, and is not hysterical. His mother and several of his maternal relatives are afflicted with some defect in the ocular muscles.

The headache from which he suffered was purely nasal in origin. He has some hypertrophic rhinitis, and the few headaches he has had since he has been under treatment have been almost immediately relieved by a reduction of the pressure from the enlarged and engorged middle turbinates. The pain has always been unilateral, and it has been entirely absent since his catarrh has been treated regularly.

When he lies flat on his back both lids remain wide open without effort, and all the muscles appear well balanced.

Notes by Dr. Risley, January 12, 1905. When first seen by us, October 28, 1904, the following conditions were present: The vision in the right eye, with his correcting glass, was 6/x; in the left eye, 6/viii. The ophthalmoscope showed the fundus oculi cherry-red and fluffy, with large central excavations in the disks, but no signs of atrophy or swelling. There was present diplopia caused by a partial paralysis of the internal rectus, and a complete paralysis of the inferior rectus on the right side. At his next visit, November 25, 1904, the double images were closer together, and a curious ptosis was present, usually affecting the left eye, but at times both eyes. *He stated that on arising in the morning the eyes were wide open, and the double images very close together, and at times he had no perceptible diplopia, but as he resumed the duties of the day all the symptoms recurred.* The left eye at this visit was slightly turned upward, apparently by the effort to overcome the ptosis. There were crossed diplopia and left hyperphoria.

Examination. Eyes left=exophoria disappears, hyperphoria increases. Eyes left and up=hyperphoria increases, slight exophoria. Eyes right and up=exophoria increases, hyperphoria disappears. Eyes right=exophoria increases and hyperphoria increases

Eyes right and down = hyperphoria and exophoria increase. Eyes down = exophoria and hyperphoria diminish. Eyes down and left = exophoria and hyperphoria diminish.

Four years ago a friend of the patient, an optician, told him he had muscle trouble when testing his eyes.

Notes by Dr. Spiller, January 10, 1905. The patient had severe headache during ten years until about six or seven months ago. Vision was not affected when he had the headache and has always been good.

When he takes off his glasses the upper lids begin almost immediately to droop and continue to do so until the eyeballs are covered, first one then the other. He wears a cover over one eye, and changes this from one glass to the other frequently. When he looks downward both upper lids fall. The external rectus on each side is normal. He cannot look upward or downward very well with the right eye, but can perform these movements with the left eye. He cannot keep both eyes open at the same time more than a minute, but if he covers one eye the other remains open. The iritic response to light and accommodation is prompt in each eye. Sensations for touch and pain are normal in all parts of the body. The muscles of the face, of the tongue, and of mastication are normal. The biceps reflex is not unusually prompt on either side. The grasp of each hand is powerful. Voluntary power in the lower limbs is normal. The patellar reflex is normal on each side. Ankle clonus is not obtained. Gait and station are normal with eyes open or closed. The muscles of the facial distribution respond promptly to the faradic current.

January 24, 1905. When he rises in the morning he cannot open his eyes so well as later in the day; formerly this was not the case. He can open his eyes better in the evening; during the entire day he uses his eyes in reading orders in his store (he is a groceryman) or in writing, therefore the disturbance is greater during the day, and is less in the evening, because he lies down.

When he takes off his glasses the upper lid of one eye, depending on which eye he had been using last, begins to fall, and gradually the upper lid falls until there is complete ptosis; while this is occurring, the upper lid of the other eye gradually droops, until ptosis may be complete or nearly complete on this side. The falling of the upper lid cannot be due to an attempt to overcome the diplopia, because the second lid droops after the first pupil is covered by the lid, and the falling of the lids occurs in the same manner when occasionally he has no diplopia while looking directly forward. He usually has diplopia in looking directly forward, but occasionally he has no diplopia for a few minutes, and then one eye "shoots off," as he expresses it, and diplopia develops. *When the upper lids have fallen, if he closes his eyes for two or three minutes he can open them again,* but with some ptosis of one lid; to-day it is in the left.

FIG. 1.

The ptosis of the right eye is not so great as that of the left. The photograph was taken one or two minutes after the glasses were removed. At times he is able to open both eyes fully.

FIG. 2.

The eyeballs are almost completely covered. The photograph was taken after the glasses had been removed three or four minutes.

He cannot keep both eyes open at the same time more than one or two minutes. If he covers the left eye with his hand, the right eye stays open better, but after two or three minutes the right upper lid falls. He now can keep the left eye partially open if he covers the right eye. The weakness of the other ocular muscles seems now to be in the elevators and depressors of the right eyeball. The ocular palsy, he says, has seldom been the same at any two examinations. The deltoids react well to a rapidly interrupted faradic current, but there seems to be some exhaustion by this current in the left sternocleidomastoid muscle.

MYASTHENIA GRAVIS:

WITH SPECIAL REFERENCE TO THE OCULAR SYMPTOMS, AND A REPORT OF A CASE INVOLVING THE EYES ONLY.[1]

By Mortimer Frank, B.S. (Mass. Inst. Tech.), M.D.,
of Chicago, Ill.

Myasthenia gravis is rare, especially in children. Of the earlier investigators, six are pre-eminent, viz., Wilks, Erb, Oppenheim, Hoppe, Goldflam, and Jolly. The disease was first described by Wilks[2] in 1877. He recorded a case of apparent bulbar paralysis, in which death resulted in a few weeks from failure of respiration. No lesion of the medulla was found at the autopsy. In 1878 Erb[3] published three cases, in two of which the principal symptoms were bilateral ptosis, while in the third, the intrinsic muscles of the eye were also involved. Weakness of the neck muscles, paresis of the muscles of mastication and weakness of the tongue and extremities were other symptoms observed. No muscular atrophy was present. Nothing more was added to the subject until nearly ten years later, when Oppenheim,[4] in 1887, published "A Case of Chronic Progressive Bulbar Paralysis without Anatomical Findings." Shortly afterward Eisenlohr[5] reported a case in an eighteen-year-old girl, in which the prominent symptoms were ptosis and paresis of other ocular muscles. In this case, as in Oppenheim's, no lesion was found. In 1891, Goldflam[6] added four new cases to those previously reported and collected by him. In 1892 Hoppe[7] contributed an exhaustive account of the subject, signalizing a new epoch in the history of the disease. Goldflam showed that the characteristic

[1] Read before the Chicago Medical Society, November 2, 1904.
[2] Guy's Hospital Reports, 1877, vol. xxii.
[3] Ueber Einen Neuen, wahrscheinlich bulbären Symptomenkomplex, Arch. f. Psych., Bd. ix.
[4] Virchow's Archiv, Bd. cviii.
[5] Neurol. Centralbl., 1887, Bd. vi.
[6] Ibid., 1891, Bd. x.
[7] Berl. klin. Woch., 1892, Bd. xxix.

muscular symptoms—fluctuating in intensity, improving by rest and becoming aggravated by fatigue and exertion—were an abnormal tiring rather than an absolute paresis. As an addition to the clinical picture Jolly,[1] in 1895, described the peculiar electric reaction of the nerves and muscles known as the "myasthenic reaction"—*i. e.*, the muscles and nerves reacting normally to electricity are rapidly exhausted by the faradic current, but respond again after rest. This electric reaction is quite variable. In some cases it is marked, in others slight. It may be present at one time, and absent at another in the same case. Again, it is often present in a certain group only of the affected muscles. The number of names by which the disease is known is numerous. Among them are bulbar paralysis without anatomical lesions, maladie d'Erb, syndrome d'Erb et Goldflam, myasthenie grave pseudoparalytique (Jolly), chronic progressive bulbar paralysis (Oppenheim). Strümpell[2] called the affection asthenic bulbar paralysis.

The most salient features of the disease are briefly as follows:

Etiology. Sex has a marked influence. The disease is most frequent in the female. Of the reported cases, there were nearly twice as many females as males. It appears most often between the ages of twenty and thirty years. Only three cases under ten years of age have been reported. One of these was a child of four years (Karplus[3]), and another of five years (Goldflam).[4] A third was a child of eight years (Gowers).[5]

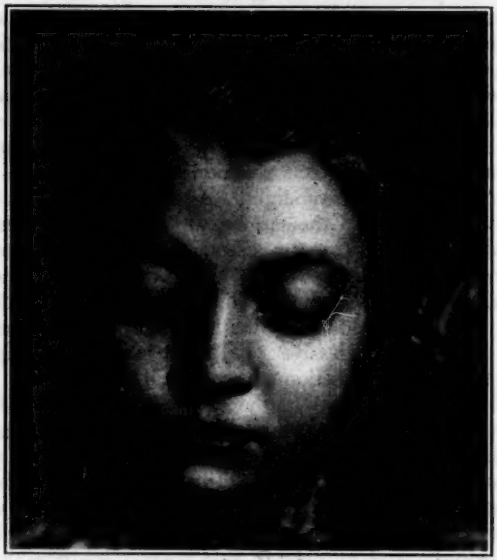
No real cause for myasthenia gravis is known. Some prostrating infectious disease, such as tuberculosis, influenza, typhoid, diphtheria, etc., frequently precedes the affection. Toxic causes, as alcohol, rheumatism, and syphilis, have been assigned, but their significance is unknown. Anæmia, menstrual disorders, and pregnancy were noted in women. Mental strain and anxiety, exertion and migraine, occasionally precede myasthenia gravis.

Onset. This is usually gradual; but occasionally the symptoms increase quite rapidly, death resulting within a few weeks. Such a case is reported by Devendorf.[6] Weeks, months, or years elapse sometimes before symptoms of the disease appear other than ocular paresis. In Karplus'[7] patient ocular paralysis occurred at intervals for nineteen years before any other signs of the disease were observed.

Symptomatology. An early and most important symptom coming on gradually and insidiously is ptosis. In nearly half the cases this was the first symptom to appear. It was present in over 80 per cent. of all recorded cases. "When it is remembered how easy it is for a patient to overlook a slight ptosis, this proportion

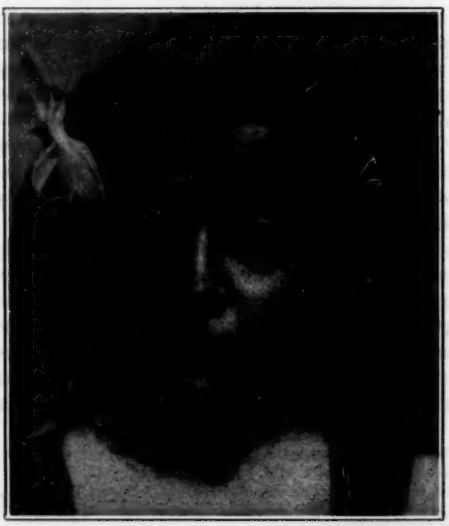
[1] Berl. klin. Woch., 1895, Bd. xxxii.
[2] Deutsch. Zeitschr. f. Nervenheilk, 1896, Bd. viii.
[3] Jahrb. f. Psych. u. Neurol., 1897, Bd. xv.
[4] Neurol. Centralbl., 1902, Bd. xxi.
[5] British Medical Journal, 1902, vol. lxxxv.
[6] Deutsch. med. Woch., 1902, Bd. xxviii.
[7] Ibid.

FIG. 1.



Position of the lids when no effort is made to raise them. Note the inability to completely close the lids.

FIG. 2.



Strong effort made to raise the lids. The absence of wrinkles in forehead is very noticeable.

seems all the more remarkable" (Hun).[1] As a rule, the ptosis is unilateral at the onset, but it soon becomes bilateral and more marked on one side than on the other, as in the case reported (Figs. 1 and 2). In the morning after the night's sleep it may not be present, but shortly after the patient rises it rapidly becomes marked, and is always worse toward the end of the day or after looking up for any length of time, when the lids gradually droop. Compensatory action from the occipitofrontalis muscle, present in other forms of ptosis, is wanting, due to its own weakness (Fig. 3). Owing to the ptosis and the weakness of the occipitofrontalis muscle the head is thrown back to enable the patient to see. The

FIG. 3.

Eyes were bandaged for ten seconds. The difference between the two eyes is well marked.

double ptosis and the effort made to see causes a sleepy appearance. Unilateral ptosis is reported by Goldflam,[2] Fajersztajn,[3] Kowjewnikoff,[4] and Raymond.[5] Complete and persistent ophthalmoplegia externa occurs without exception at some period of the disease. Although the ocular paralysis affects the extrinsic muscles of the eye, the intrinsic muscles are never paralyzed. Reaction of the pupils to light and accommodation always seems to be normal. In a doubtful case reported by Brissaud and Lautzenberg[6] the

[1] Albany Medical Annals, January, 1904.
[2] Deutsch. Zeitschr. f. Nervenheilk, 1893, Bd. iv.
[3] Neurol. Centralbl., 1896, Bd. xv.
[4] Deutsch. Zeitschr. f. Nervenheilk., 1897, Bd. ix.
[5] Gaz. des hôpitaux, 1900, tome xxi.
[6] Arch. gén. de méd., Mars, 1897.

light and accommodation reflexes of the pupil were abolished. Inequality of the pupils has been noted, and in a case reported by Buzzard[1] the pupils showed a tendency to oscillatory movements after prolonged convergence. There is frequently an inability to completely and firmly close the lids. This is present in the case herewith reported (Fig. 1). Owing to the weakness of the orbital muscles, diplopia is often present. This is associated rarely with irregular nystagmoid movements, produced by lateral conjugate deviation of the eyes. Strabismus is often present (Fig. 4). A slight prominence of the globe has been observed in a number of

FIG. 4.

Showing the divergence of the right eye.

cases, probably due to the weakness of the recti and the orbicularis palpebrarum muscles. Exophthalmos with symptoms of Basedow's disease was present in cases reported by Jendrassik,[2] Goldflam,[3] Karplus,[4] Oppenheim,[5] and others. The muscles of mastication are very commonly implicated, so that eating is a slow and difficult process from their rapid exhaustion. The muscles of deglutition are often involved, so that food collects in the roof of the mouth and is swallowed with difficulty, and liquids regurgitate through the nose, owing to the weakness of the palate. Choking while

[1] Transactions of the Ophthalmological Society of the United Kingdom, vol. ix.
[2] Arch. f. Psych., 1886, Bd. xvii. [3] Ibid. [4] Ibid.
[5] Monograph, Die Myasthenische Paralyse, Berlin, 1901.

eating is very likely to occur, on account of the diminution in the pharyngeal reflex. Weakness of the pharyngeal muscles is uncommon, but has been observed with the laryngoscope in the cases of Hoppe,[1] Buzzard,[2] and Oppenheim.[3] The speech, at first normal, shortly becomes nasal and the words grow more and more indistinct, until there is complete aphonia. Neither muscular atrophy nor the reaction of degeneration is ever present. Pain is a rare symptom. The muscular symptoms are, as a rule, bilateral. The weakness of the muscles results in their rapid exhaustion upon active movements. Bulbar paralysis is present to a greater or less degree, and finally dyspnœa after exertion is a striking symptom. From this the patients usually die. The phenomenon of muscle tiring and permanent paresis is found in those voluntary muscles which are continually in tonic contraction and have little rest, such as the levator palpebræ, the muscles of expression and mastication. The purely reflex acts of the eye and extremities do not show any abnormal tiring, Jolly,[4] Buzzard,[5] Campbell,[6] and others. This phenomenon of weakness, as a rule, is not present in all of the muscles, but is limited to a few muscles or a group of muscles, as in the case reported. This particular symptom may wholly and unexpectedly remit for several days. Indeed, remissions and exacerbations are the rule, but the weakness is always worse in the evening.

Pathology. The pathology is vague and uncertain. In the autopsies that have been made there is no evidence that the disease is associated with any lesion of the nervous system. In over half the cases no lesion was demonstrable. Many hypotheses have been brought forward in explanation of the symptoms, but as to the exact nature of the disease we are profoundly ignorant. From the clinical conditions, gross lesions could not be expected.

Prognosis. Judging from the reported cases, the prognosis as regards recovery is practically hopeless. Apparent recovery has been noted, but when we recall the great improvement and apparent recovery lasting weeks, months, or years, and followed by a relapse we must look upon permanent recovery as doubtful. The average duration of the disease is from one to three years. Of the one hundred and twenty cases referred to fifty died.

Diagnosis. The diagnosis is easy in typical cases when the rapid tiring and variation in the intensity of the symptoms with the myasthenic reaction appears; but the diagnosis is especially difficult, and at times impossible, in the early stages of slowly developing and atypical cases. The possibility of hysteria being mistaken for this disease is great, especially when the only symptoms are ptosis

[1] Monograph, Die Myasthenische Paralyse, Berlin, 1901.
[2] Ibid.
[3] Ibid.
[4] Ibid.
[5] Ibid.
[6] British Medical Journal, 1899, vol. i.

and diplopia, appearing and disappearing without apparent cause. Ocular paralyses, rapid tiring of the muscles are rarely found in hysteria, while the myasthenic reaction is not. As in other hysterical paralyses, those of the eyes are characterized by their lack of permanency, their change under psychic influences and their association with sensory disturbances and other hysterical stigmata. In the early stages of the disease it might readily be mistaken for nuclear ophthalmoplegia, and when the ophthalmoplegia alone is present, the diagnosis is extremely difficult. In myasthenia gravis the muscles regain a certain amount of power after rest, but sooner or later the other symptoms of the disease appear. In pseudobulbar paralysis the ocular muscles are not involved, and the myasthenic reaction and rapid tiring are not present.

TREATMENT. The treatment consists first in absolute rest, gentle massage, and measures to maintain a high standard of nutrition. A mild galvanic current has been used, but its benefits are questionable. Many drugs have been tried, but none seems to have any favorable influence over the progress of the disease. The case about to be described illustrates very nicely the ocular phase of the disorder.

The patient, E. M., a female, aged ten years, of American birth, but of Bohemian parentage, was referred to me in November, 1902. At the time of my first examination there was present double ptosis, most marked on the right side (Figs. 1 and 3), paresis of all the extrinsic muscles of both eyes, and a marked divergent squint of the right eye (Fig. 4), with complete preservation of the functions of the intrinsic muscles. The iris reacted to both light and accommodation. The fundi were normal, and save for an error of refraction—O.D.+1.50—3.50 X 180° V. 6/9+: O.S.—1.25 X 180° V. 6/5—the visual faculty was normal. Her history is as follows:

In 1898, when the patient was four years old, she was ill, but nothing further can be learned. Two years following, in 1900, her mother first noticed a drooping of the right eyelid, and a few weeks later the left became affected. She was taken to the Illinois Charitable Eye and Ear Infirmary in November, 1901, and all that the records there show is the following:

"Case No. 108,110, E. M.; mixed astigmatism; ptosis acquired one year; defective motion of all external eye muscles except left external rectus." The child is well nourished, and repeated examinations show nothing abnormal with any other functions of the body. From the beginning there was no evidence of ptosis upon getting up in the morning, but gradually the drooping becomes marked as the patient gets about. Early in the history the ptosis disappeared entirely for a day or two, only to return again. The eyelids can be closed, but not tightly, and, owing to the weakness of the occipitofrontalis, the forehead can be moved but very little. The patient tries to overcome the ptosis by throwing her head back.

With the persistence of the ptosis there was observed a gradual progressive impairment of power in the movements of the eyeballs, but without diplopia. This increased until the eyes became completely fixed in the primary position. At the present time there is about one millimetre of lateral excursion in either eye, but motion is barely perceptible in the vertical. If the eyes are bound lightly with a bandage or even kept closed for a few seconds, the movement of the lids appears normal (Fig. 2), but raising the lids three or four times in succession causes complete exhaustion. The movement of the globes improves only slightly with rest. A feeble myasthenic reaction has been demonstrated at times, and when sought for again was entirely absent. There is no history of tuberculosis, syphilis, or nervous disease in the family. There is nothing abnormal in the motor or sensory distribution of the fifth nerve.

Let us now consider what the nature and seat of a lesion would probably be that would cause an impairment of function of part of the third nerve and also the fourth and sixth on the same side, and do no damage to the structures in the immediate vicinity. An intraorbital lesion of the third nerve, or one involving its intracranial trunk, would not account for it, for the ciliarius or the sphincter iridis would not escape. Could a gross lesion, such as a tumor, meningitis or an area of softening, affect the nuclei of the third, fourth, and sixth nerves, and at the same time not involve the centres for the iris and ciliary muscle, the fifth and seventh or other neutral tracts? To this question we must answer no, excepting that we assume several independent lesions have involved the nuclei of a set of associated nerves on both sides, which is quite improbable. The most rational hypothesis, then, is that the disease is purely a motor one, and that the lesion must be situated either in the muscles themselves or in the motor nuclei. It has been demonstrated by Hensen and others that the innervation of the sphincter of the iris and the ciliary muscles arises from nuclei separate and distinct from those giving origin to the remaining parts of the third nerve. The myasthenic reaction, and the nature and distribution of the symptoms, point to a disturbance in the muscles or the peripheral motor neurons, rather than to a lesion in the motor nuclei. Whether this can be explained as due to the action of a toxin powerful enough to produce symptoms of the disease, and yet produce no demonstrable structural changes, or to lymphoid infiltration of the muscles as found by Weigert,[1] Goldflam,[2] Link,[3] and Hun,[4] is a question unsettled, as yet. Until some definite and characteristic anatomical lesions are proven, we can only theorize.

[1] Neurol. Centralbl., 1901, Bd. xx.
[2] Ibid.
[3] Deutsch. Zeitschr. f. Nervenheilk., 1902, Bd. xxiii.
[4] Ibid.

MYXŒDEMA FOLLOWING EXOPHTHALMIC GOITRE.

By N. B. Foster, M.D.,
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From time to time there have been reported cases of myxœdema which either preceded or followed upon exophthalmic goitre. The following case is one in which, to use the terms of Moebius and Greenfield, athyrea succeeded upon hyperthyrea. The notable feature of this case is the remarkable amount of gastric disturbance that the patient has suffered, and, to a less extent, still experiences; otherwise there can be claimed nothing peculiar; similar cases have been reported by Kraft-Ebing, Felin, Fauer, and Osler.

Rebecca L., aged fifty-seven years, married, housewife, was admitted to the New York Hospital December 11, 1903; service of Dr. Peabody. The patient complained of nervousness and weakness.

Family History. There is no history of tuberculosis or cancer. None of her relatives has had joint diseases. Her parents, brothers, and sisters are free of any nervous troubles, and she knows of no one in her family that ever had a goitre.

Past History. She does not remember any disease in childhood. She had typhoid fever "many years ago," but has never had chills and fever, pneumonia, nor pleurisy. She has had various rheumatic "twinges," but has never been sick in bed with joint pains. She has never been jaundiced. There is no history of chronic cough or of any symptoms of pulmonary disease. The patient states that she has not suffered from shortness of breath in past years, and her ankles have never been swollen; her appetite has been moderate, food never causing distress; bowels usually regular.

The menses have been regular, but always attended with some pain. She has been married a number of years (cannot recall the year of marriage), but has never had a child. There have been no miscarriages. The patient was operated upon at the Roosevelt Hospital in 1901 for uterine fibroids, the uterus being removed, but not the ovaries.

Present Illness. The patient affirms that aside from always being very nervous, she has enjoyed good health until the onset of the present affection. In 1887, after returning from South America, she noticed that her neck was considerably swollen. This increase in size continued for some time; she is not sure how long. She was then told that she had a goitre. At this time the patient says she was "horribly" nervous, her hands shaking violently, and she suffered from palpitation of the heart. Her friends often told her that her eyes were "swollen." She thinks that she continued in this

condition without much if any change until six months previous to entering the hospital. Over a year ago, however, she says that she noticed that her skin was very dry and came off in fine scales, and that her hair was dry and becoming thin rapidly. A leukodermic spot on her hand was much more marked then than at present.

About six months ago the patient says the palpitation of the heart became considerably aggravated, and that she had dull, aching pains in her legs. At about this same time her body began to swell, and she became "puffy" all over. Her strength was so lacking that she could not leave her bed, and there she remained for seventeen weeks. During this time she was utterly wretched from the nervousness and the aggressive sense of weakness, and frequently meditated on self-destruction. Her memory was very defective then, she thinks, and is now, since she recalls events slowly and with evident effort. While in bed she vomited frequently, lost her appetite, and ate only when urged to do so. She says she vomited several times every day, and that some degree of nausea was constant. The vomiting had no relation to the taking of food, the vomited material consisting usually of clear mucus, which had an extremely offensive odor. The patient states that this was so disgusting a feature that it was necessary to place her in a room remote from others in the family. The vomitus was always large in amount, on one occasion being so much during the course of the day that the attending physician measured it, and told her it was nine pints. This symptom gradually abated, but she gained no strength. During this seventeen weeks in bed she had no pain, her suffering being purely nervousness, an oppressive sense of impending misfortune, and complete prostration from weakness.

Her general condition improved, however, and, as her physician gave her no hope of further help, she came to the Out-patient Department of the New York Hospital, where she was treated with thyroid extract during the two weeks previous to admission to the wards. There appear no illusions or mental defects in the patient at present, other than slowness of cerebration and defective memory mentioned above.

Physical Examination. The patient has rather a bloated appearance, and the flesh is soft and flabby. She does not look anæmic. Her hair is quite thin and feels dry and harsh. The skin is dry, and on the body a fine, branny dust can be removed. The eyes appear normal. There is a slight tremor in the muscles of the face, and some pulsation of the veins of the neck. The thyroid gland cannot be felt. The heart is normal.

Pulse is regular, 100 to the minute, good size, moderate force and tension; the artery wall is not thickened. The lungs and abdominal organs present nothing abnormal.

Extremities. There is slight œdema of the feet; the knee-jerks are exaggerated; there is marked tenderness to pressure over the

course of both sciatic nerves. The axillary and inguinal glands are not palpable. The urine and blood are normal.

The patient was given 5 grains of thyroid extract daily, in addition to a general tonic treatment of stomachics, cold packs at night, etc. Her improvement was not marked at the end of two weeks, and the thyroid extract was doubled in dose, which caused some diminution of the nervous symptoms. A dose of 15 grains of thyroid daily was tried, but caused a marked increase in the patient's irritability and cardiac palpitation, on account of which the 10-grain dose was resumed. Improvement was very slow, but the patient was able to leave the hospital after two months (February, 1904) much less nervous and decidedly altered in appearance, the bloated, stupid look in the face having quite disappeared. She was, however, far from well, being mentally depressed and without energy.

June 15, 1904. There is no evident change in the patient's condition; she is still nervous, weak, and mentally depressed. She does some of the household duties, but says they quite exhaust her. She is still taking 10 grains of thyroid extract daily. Her general nutrition is good; hæmoglobin, 90 per cent.; urine, normal.

September 20th. Since last seen the patient has suffered several attacks of diarrhœa. She says she has pains in her abdomen every few days. When these pains are quite severe it marks the commencement of a diarrhœal attack. Her general condition, nutrition, and mental tone are not changed.

Physical examination is quite negative. She now has the harassed look of an individual suffering from one of the milder forms of insanity, and she says she is constantly worried, though she knows no cause for anxiety. Her sleep is disturbed, and nights pass when she does not sleep at all. An endeavor has been made to regulate these nervous phenomena by means of tonics and sedatives, but with no apparent success.

What may be the state of the gastric secretion, we can only conjecture, as attempts to recover a test-meal have been futile; the stomach tube causes so much distress to the patient that the experiment has been abandoned.

It appears at present that this patient is as well as she ever will be, and that she is doomed to drag out a wretched existence, unless some kindly acute infection releases her.

These cases of myxœdema following exophthalmic goitre are rare enough always to excite our interest. It is evident from cases like the above that in this type of myxœdema there is another element besides deficiency of thyroid function, or else the administration of the gland extract would produce better results. It may some time be shown that there is a perverted function as well.

THE RESULT OF SPLENIC REMOVAL:

WITH THE REPORT OF THE SUCCESSFUL REMOVAL OF A WANDERING SPLEEN WITH A TWISTED PEDICLE, OCCUPYING THE LEFT ILIAC REGION, WITH PERISPLENITIS AND NECROSIS OF THE PULP.

By James W. Hunter, Jr., M.A., M.D.,
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The spleen, like the stomach, has generally been considered a vital spot of the human economy. It was formerly taught that the loss of either meant instant death. But during the last few years we have seen the removal of both, and we are led to believe that neither is essential. The digestive function can be entirely assumed by the intestine, and, though a frequency of eating be demanded, there is no impairment of health. Whatever the function of the spleen, whatever the compensatory changes when removed, we are beginning to realize that in many cases a splenectomy is not only justifiable, but eminently to be sought. It is in this connection that I wish to report a case of wandering spleen with twisted pedicle, occupying the left iliac region, a perisplenitis, and a necrosis of the pulp.

On account of the close anatomical relation which the spleen bears to the stomach, it was but natural to suppose that in some way the organ was concerned in digestion or metabolism. Along this line there have been many investigators. No suitable explanation has, however, been offered for the splenic enlargement occurring in most of the acute infectious diseases. Whether the spleen plays any role in the production of toxins or antitoxins is not known. Yet, Courmont and Duffon[1] found that spleenless animals resist diphtheria better than normal ones, and the more recent the removal the more marked the resistance.

Noël Patton,[2] after an extensive series of experiments upon the spleenless dog, concludes (1) "that there is a more rapid excretion of water after a meal, probably indicating a more rapid absorption," and (2) "that there is no essential difference in the course or nature of the metabolism either during fasting or after feeding with the ordinary proteids of flesh, with vegetable food such as oat-meal, or with food rich in nucleins such as thymus gland." The later work of Fourin[3] confirms this. He removed both the spleen and stomach, and finds that, while there may be a temporary derange-

[1] Archiv. de méd. expér. et d'anat. path., Mai, 1898.
[2] Journal of Physiology, 1900, vol. xxv. p. 442.
[3] Comp.-ren. des séances et mém. de la Soc. de biol., 1902, tome iv. p. 418.

ment of the digestion due to the removal of both spleen and stomach there is no permanent interference.

The formation of uric acid has been ascribed to the spleen. Lo Monaco,[1] however, found that the output of uric acid in a spleenless patient fed on a mixed diet was approximately normal; and in no case was it noticeably diminished. Similarly, the work of Mendel and Jackson.[2] To quote: "Our experiments demonstrate that the spleen is by no means the chief organ involved in uric acid production, if, indeed, it normally plays any part whatever in the process. It seems improbable, therefore, that the formation of uric acid in the mammalia can be assigned at present to any definite organ or group of organs." My own case illustrates this also. Though no special examinations were made of the patient's urine, there was no reason for suspecting the slightest abnormality.

What then is the splenic function? Let us turn to its histological structure. Without going into details, we shall remember that it is composed of a reticular network filled in with both red and white blood corpuscles, as well as some large amœboid cells (the so-called spleen cells), and that surrounding the terminal arterioles there are masses of true lymphoid tissue (the Malpighian bodies). Bland Sutton[3] remarks that the spleen is to be "regarded as a highly developed and specialized lymph gland." If one kidney be removed the other will assume the work of both and may or may not hypertrophy. So it appears that if the spleen is removed the lymphatic glands perform its duties. There is no constitutional disturbance, and the only clinical data to be observed is a change in the structure of the blood. Thus it is proved that the splenic function deals with the blood, but just how is as yet unanswered. Many have supposed that the spleen is in some way a numerical regulator of the corpuscles, and perhaps destroys the broken-down erythrocytes. But this, with our present knowledge, can neither be affirmed nor denied.

The work of Vincent[4] is conflicting. Examining the anatomical changes in the spleenless dog, he finds in one (that furnished him by Professor Shafer, fifteen months after operation) no increase in either the size or number of the ordinary lymphatic glands, but an increase in the hæmolymph glands, "the amount of hæmolymphatic structure being decidedly greater in this spleenless dog than under normal circumstances." But no such change was noticed in five other spleenless dogs. Indeed, to quote his own words, "there was no apparent change in the lymphatic glands of any part of the body, neither in the direction of hypertrophy nor increased redness. On microscopic examination the hæmal lymphatic glands presented the features of those in a normal dog. The

[1] Bull. della Soc. Larcisiani degli Ospedali di Roma, 1894, vol. xiv. p. 102.
[2] American Journal of Physiology, 1900, pt. iv. p. 168.
[3] Lancet, January 16, 1897, p. 174.
[4] Journal of Physiology, 1900, vol. xxv. p. 2.

red bone-marrow appeared to have its usual structure." He could not say whether this was increased or not.

The work of Warthin[1] upon the sheep seems to agree with Vincent's first results. He describes a "hyperplasia of existing lymphoid tissue, transformation of hæmolymph nodes into ordinary lymphatic glands, and a new formation of hæmolymph nodes out of lobules of fat tissue, and a later proliferation of the red marrow." He finds no evidence of the formation of red blood cells in the lymph nodes after splenectomy. "The function of hæmolysis is taken up first by the hæmolymph nodes, later by the ordinary lymphatic glands." Continuing, he remarks that "the hæmolytic function of the hæmolymph nodes and hyperplastic lymph glands exceeds that of the primitive spleen, causing an excessive destruction of red cells. The resulting anæmia is later compensated for by an increased activity on the part of the bone-marrow. It would appear, therefore, that the removal of the spleen leads to an increased production or retention of some hæmolytic agent usually disposed of by the spleen. The effect of this hæmolytic agent is either to stimulate the phagocytes in the hæmolymph nodes to increased activity, or to change the red cells so that they are more easily destroyed by these phagocytes." "The presence," he concludes, "of great numbers of eosinophiles in the glands, showing great destruction of red cells, seems to point to some relationship between these cells and hæmolysis." He confirms the appearances described by Tizzoni, Wimgradow, Eterod, Griffin, and Mosler, but interprets them differently. He also confirms Bayer's work upon the regeneration of the lymph glands.

Thus it is conclusively proved that the lymphatic glands take up the work of the spleen. These may or may not enlarge or undergo a change; different species seem to react differently, or even individuals of the same species. In human beings the result is still doubtful, although Mendel and Jackson state that an enlargement of the lymphatic glands has been recorded after splenectomy. Bayer[2] declares that enlargement of the thyroid and lymphatics occurs in many cases, though transitory. But I would mention that a most careful examination of the cervical glands, thyroids, and tonsils of my own patient showed no abnormality. What compensatory changes occurred in the mesenteric glands, I am, of course, unable to say.

According to Bayer, the immediate results of splenectomy are an increase of the leukocytes, a decrease of the erythrocytes, and a reduction of the hæmoglobin. These and the changes noted above are only transitory; in the course of a week or month the blood will become normal. Cabot[3] states that after splenectomy from

[1] Contributions to Medical Research, Ann Arbor, 1903, p. 234.
[2] Münchener medizinische Wochenschrift, Bd. li. p. 116.
[3] Clinical Examination of the Blood, New York, 1904, p. 181.

whatever cause there is a lymphocytosis. "Later, after many months, a moderate eosinophilia may appear and the mast cells are increased." In 3 cases cited by him the white blood count ranged from 17,500 to 27,000 in from five weeks to two months after operation. In 21 cases collected by Stahelin[1] there was a leukocytosis varying from 12,000 to 60,000 per cubic mm., according to the length of time, usually from 10,000 to 17,000.

Rautenberg,[2] so far as I can observe, has published the best set of observations. His case showed immediately before operation some 5000 white blood cells. Three days afterward it had risen to 6500, and gradually increased until the thirtieth day, when a maximum of 10,000 was attained. There was then a gradual fall, until nine and a quarter months after operation, when it reached 5300, only to rise later to 6500. The reds rose from 4,310,000 to 5,100,000, attaining a maximum at the same time as the whites, and falling within ten months to 3,750,000. In the meanwhile the lymphocytes had increased from 19.3 per cent. to 33.4 per cent., when they, too, began to fall; the eosinophiles reached 8.8 per cent. All these changes, except the last one, occurred when the leukocyte count was the largest, some thirty days after operation.

Stahelin's case, like my own, was complicated by an abscess. His counts, made from two months to fourteen months after operation, showed changes similar to Rautenberg's, the blood finally becoming almost normal. Below will be found some comparative counts.

	Reds.	Hæmoglobin.	Whites.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Basophiles.	Eosinophiles.
Rautenberg:								
Before operat'n	4,310,000	...	5,000	75.6	19.3	1.6	1.3	2.2
30 days after	5,100,000	...	10,000	62.0	31.8	0.4	0.8	5.4
9¼ months	3,720,000	...	5,300	61.2	28.4	1.2	2.0	7.2
Stahelin:								
2 months after	4,700,000	...	15,700	34.2	59.1	2.2	0.3	4.2
6½ months	3,500,000	...	10,000	67.9	26.7	1.8	0.1	3.5
14 months	5,300,000	...	7,500	58.5	35.9	1.75	0.15	3.7
Webster:[3]								
6 days	4,300,000	60	22,000	77.0	14.0	8.0	0.0	2.0
26 days	4,420,000	...	11,600	65.2	12.0	18.8	0.0	2.0
My own case:								
6 weeks after	5,000,000	95	14,400	43.0	42.3	10.7	1.5	2.5

The fever after a splenectomy runs no special course. It is only necessary to remember that in the weakened state of the body any latent or intercurrent disease (such as malaria) will develop. Rou-

[1] Deut. Archiv f. klin. Med., 1903, p. 364.
[2] Münchener medizinische Wochenschrift, 1903, No. 16, p. 684.
[3] Journal of the American Medical Association, 1903, vol. xl. p. 887.

tier,[1] of Paris, reports the removal of a spleen weighing 3080 grams (six and three-fifths pounds). There was no histological abnormality, but, as the patient had a chill followed by a temperature of 40° C. (104° F.) every five days, he concluded that he was dealing with a malarial (paludique: "salt water") enlargement. I have been unable to refer to Moraczewski's paper[2] on the course of fever after a splenectomy, but would say that in my own case I could find nothing not better explained upon other grounds. The development of an intermuscular abscess caused a rise of temperature as high as 104.4° F. (40.2° C.) on the fifth day, which gradually subsided after a thorough drainage. A secondary rise of temperature yielded to quinine, and I would conclude, like Routier, that a latent malaria was present, especially as the patient was from a malarial region. Aside from this nothing abnormal was noted.

The statistics of splenectomy show a constant improvement. Thus, to quote Bayer: "Bessel-Hagen reports up to the year 1900 a total of 358 cases of total extirpation of the spleen, which does not include those spleens projecting through a peritoneal wound (according to Jordon 28 in all), and which were removed with cure. Of the 358 cases of Bessel-Hagen there were 225 cures and 133 deaths. The mortality is then 37.2 per cent., whereas in the six earlier years it was, according to Vulpius, 49.6 per cent., and according to Ceci, 51.6 per cent. The difference in these statistics, resulting in only six years, shows a great improvement. But Bessel-Hagen says that if those cases were omitted which to-day would be considered inoperable, the percentage of deaths would be reduced, namely, 27.6 per cent. deaths. He goes farther and compares those cases which were operated upon up to the year 1890 with those until 1900. Whereas it was at first 42.2 per cent., it was for the years 1891 to 1900 only 18.9 per cent. Moreover, it is interesting to note that Bessel-Hagen does not consider 10 cases of malarial hypertrophy, which befel one surgeon (Jonesco) and which were moribund, as well as a case of idiopathic hypertrophy whose disastrous result was due wholly to a mistake of the operator, as unavoidable. But if we consider the admission of these 11 cases as arbitrary and therefore inadmissible, there is a reduction of the mortality from 42.2 per cent. during the time up to 1890 to 26.3 per cent., including the years from 1891 to 1900."

Osler,[3] on the other hand, in the 1902 edition of his work, reports (on the authority of Warren) a total of 43 splenectomies for myelogenous leukæmia, of which only 5 recovered. "Fussell," he says in the 1900 edition, "gives the statistics of 105 cases of splenectomy with 48 deaths. Of the cases of simple hypertrophy, 28 in number, 9 recovered. Of 16 cases of floating spleen, 15 recovered." Bayer

[1] Bull. et mém. de la Soc. de chir. de Paris, 1904, p. 366.
[2] Gaz. lek. Warszawa, 1902, q. s. xxii. p. 868.
[3] Principles and Practice of Medicine, New York, 1900 and 1902.

himself gives a series of 27 wandering spleens with only 2 deaths, while Bland Sutton succeeded in collecting a score of cases operated on within the years 1877 to 1897 without a death. Thus it would seem that, with properly selected cases, the recoveries after splenectomy should equal those of any other abdominal operation.

On August 30, 1904, Mrs. E. consulted me, complaining of a tumor in the left iliac region. She had noticed a small tumor some seven years ago, frequently changing its position, but as it gave no trouble no particular attention was paid to it. It had, however, gradually enlarged. About four months prior to her visit she had strained herself while picking strawberries, and had had frequent attacks of pain in the region of the tumor, which meanwhile had assumed a rapid growth. An examination showed an elliptical tumor in the left iliac region, some soreness, and a slight fluctuation. A later vaginal examination gave no additional evidence. The patient then stated that the tumor had at one time occupied the right iliac region, and that in her youth she had had a "spleen," which, however, disappeared after treatment. Whether this had anything to do with the later developments I cannot say, but it does not seem improbable.

Her family physician, Dr. J. M. Newbern, writes that Mrs. E. was married at sixteen, and has had five children (the youngest three), but no miscarriages. Her health had been fairly good until some nine months before her recent attack, on June 24th, though previous to her marriage she had suffered with malaria and chlorosis. Enjoying a good appetite, she would sometimes eat too heartily, with a resulting attack of indigestion; no pain in the abdomen, but a severe one just under the left scapula. "I called to see her on June 24th," continues Dr. Newbern, "and found her with a high fever, intense pain in the left iliac region, and also a hard tumor. It was so painful that I could not examine her carefully. Her bowels were constipated, and it was with much difficulty that I got them opened."

At the time of the operation it was noticed that the tumor had moved nearer the midline, but I judged that its altered position was due to the emptying of the bowel. Upon opening the abdomen, I found a large brown mass of a semisolid consistency bound to the abdominal wall. It appeared so likely that I was dealing with some kind of a cyst, that a trocar was inserted, but with no result. The anterior adhesions were broken up, only to find that still stronger adhesions bound the tumor to the intestine. These were in turn torn apart and the pedicle ligated. But before doing so it was discovered that the pedicle was twisted and the splenic artery completely occluded. The abdomen was then washed, filled with sterile water, and closed.

Mrs. E. remained in the hospital some six weeks. Aside from the development of an intermuscular abscess (and a consequent

fever) the convalescence was almost uneventful. A second rise of temperature yielded to quinine, probably showing a latent malaria. An attack of intestinal indigestion toward the end of convalescence caused some disturbance; this, however, was not repeated. Bland Sutton states that it is a fact that many if not all individuals with wandering spleens are very thin and often gaunt, and so far as his observations go they retain this character when spleenless. Mrs. E., on the contrary, while a small woman, showed no signs of emaciation, and a letter from her husband a month after her return, assures me that she has gained in weight twenty pounds. Six weeks after her return she weighs one hundred and fifteen pounds, assists Mr. E. with his business, and is getting fat.

The tumor presented an elliptical appearance, being 19 cm. (seven and a half inches) in length, by 10.5 cm. (four and a quarter inches) broad, and 10.0 cm. (four inches) in thickness, of a gelatinous consistency, and dark brown in color. The capsule refused to strip, was greatly thickened, and measured 1 cm. On cross-section several soft spots were found near the hilum. These appeared necrotic, easily washing away. The pulp itself bulged greatly, showing that the capsule was under considerable pressure. The weight of the spleen is variously given by the different authorities as from 150 to 250 grams. This one weighed 700 grams (one and a half pounds). It was removed none too soon.

The microscopic examination revealed several old hemorrhages, as well as a large amount of pigment scattered throughout the mass. The sloughing pulp proved to be necrotic, the tissue refusing to take a basic stain, and staining but poorly with eosin. No nuclei or leukocytes were found; the Malpighian bodies were scarcely visible. The investing envelope showed a division into three layers; an outer fibrous (formed from the peritoneal attachment), a mass of dense fibrous tissue (the original capsule with some hypertrophy), and a third formed of a greatly enlarged and dense network (the old trabeculæ) filled in with young connective tissue. In other words, there was a subacute inflammation of the capsule, a chronic passive hyperæmia of the pulp, and a well-advanced necrosis. So far as I know, there is no description of this method of capsular hypertrophy in our text-books, and it therefore seems well to lay a special emphasis upon the condition here noted.

A careful examination of all the available literature since 1890, while showing perhaps 150 removals, gives only 22 operations on cases of wandering spleen. Of these only 8 (the cases of Sutton,[1] 1892; Hertaux,[2] 1893; Malin,[3] 1894; Runge,[4] 1895; Chandleux,[5]

[1] Transactions of the Clinical Society of London, 1892–1893, vol. xxvi. pp. 46–49.
[2] Bull. et mém. de la Soc. de chir. de Paris, 1893, n. s. tome xix. p. 752.
[3] Lancet, London, 1894, vol. ii. p. 627.
[4] Berl. klin. Wochenschr., 1895, Bd. xxxii. p. 346.
[5] Bull. de la Soc. de chir. de Lyon, 1899–1900, tome iii. p. 92.

and Stengel,[1] 1899; Bennett,[2] 1900, and Webster,[3] 1903) had a twisted pedicle. Of these 8, Webster's and Bennett's cases were attended with a necrosis, though only one (Webster) mentions an adhesion. But assuming that several of the 8 reported since 1890 were complicated by adhesions or necrosis, my own case is the ninth.

My thanks are due to Dr. J. M. Newbern, of Jarvisburg, N. C., for the history of the case; to my associate and consultant, Dr. H. M. Nash, of this city; and to Drs. H. S. Baker and W. C. Campbell, of the Norfolk Protestant Hospital, for their many courtesies.

RECENT ADVANCES IN THE TECHNIQUE OF ROENTGEN-RAY THERAPY.[4]

By Charles Lester Leonard, A.M., M.D.,
of Philadelphia.

The great variance in the results reported in the treatment of disease by the Roentgen method must strike the reader of medical literature very forcibly and lead him to wonder if there is any truth in the favorable reports, or any logical explanation for the diversified and often apparently antagonistic physiological effects.

This feeling of doubt is readily enforced if he has chanced to come in clinical contact with the results of an inexperienced operator. It must, however, be remembered that positive evidence is always more valuable than negative. That even a few accurate observations of beneficial results attained are far weightier evidence of therapeutic efficiency than any number of failures. The time has not yet arrived when cures can be demonstrated; a longer period of therapeutic employment is necessary, though many cases have remained apparently cured for over two years. An inhibitory action has, however, been absolutely demonstrated. The power to inhibit the growth of malignant or other pathological cells has been demonstrated beyond question. This agent has been proved to be more potent than any other agent heretofore employed in checking the growth of malignant cells. Even operation, which was the most effective method of treatment, does not inhibit the growth of disease; it eradicates it by removing it from the body, but in no way alters the nature or character of the cellular growth.

[1] American Surgery, Philadelphia, 1899, vol. xxx. p. 321.
[2] Texas Medical News, Austin, 1900-1901, vol. x. p. 703.
[3] Journal of the American Medical Association, Chicago, 1903, vol. xl. p. 887.
[4] Read before the College of Physicians of Philadelphia, February 1, 1905.

Operation is not curative except by being destructive; the Roentgen ray has been demonstrated to have inhibitory power over the cellular malignant activity of new-growths; it is reconstructive rather than destructive.

While the failures to secure permanent results far outnumber the successes, they do not outweigh them. The Roentgen method has been too frequently asked to accomplish results that were impossible by other means, and hopeless cases have been the rule rather than the exception. When it has had the opportunity to co-operate in treating cases after surgical removal of the focus of malignant disease, the results obtained in the treatment of these conditions will be improved. Roentgen treatment should supplement but never supplant operation, where operation is admissible. But to attain the highest degree of success, cases must be sent early for Roentgen treatment, as well as early for operative intervention.

The apparent variation in the effect of the Roentgen rays on disease is to be explained by differences in the technique employed by operators, and by inefficient employment resulting from inexperience and the timidity which it breeds.

The prevailing idea that this method of treatment is successful in and can only hope to influence superficial malignant growths is erroneous. The rarity of cases in which it has affected deep-seated growths has been due to lack of knowledge of the qualities of Roentgen rays suitable for their treatment and defective technique and experience in employing them. There are enough authenticated cases to demonstrate its power, and recent advances in the knowledge of technique has placed them more readily under the influence of this agent.

The facts already demonstrated make it the most potent alterative and curative agent known for the treatment of malignant disease. They make it imperative that every conscientious practitioner should carefully consider the employment of this agent as a supplementary method to surgical operation in the treatment of every case of malignant disease that comes under his care. The mortality from malignant disease even after early radical surgical removal is so great that so powerful a supplementary method of treatment ought not to be neglected.

A review of the probable physiological action and pathological changes that follow its employment will in a measure explain the rationale of its wide field of application and harmonize its apparently antagonistic effects. A study of the principles that underly its application will help to a realization of the difficulties that must be mastered by technique before accuracy and efficiency in dosage can be expected. The variation in results can then be more readily understood and can be shown to follow defective technique and not to be due to the valuelessness of the agent. It will be easy to

appreciate why results can be obtained by those possessing technique and experience, while others fail even to improve similar cases.

A reasonable deduction regarding the physiological action of this agent can be gained from the clinical observation of its effects. Stimulation of nutrition, both local and systemic, has been almost universally observed where physiological effects have been produced. Changes due to altered metabolism are seen in all cases and pathological examinations have repeatedly demonstrated varying degrees of retrograde metamorphosis in tumors that have been effectively treated. This stimulant action when pushed produces decided alterative effects, which have been ascribed to the autolytic ferments within the cells and results finally in their dissolution and absorption. The effect of these forces, fortunately, is varied in its action on normal and pathological cells, a result not uncommon to stimuli. The stimulus which will injure pathological cells has no harmful effect upon normal metabolism, but rather increases the vitality and growth of normal tissues. This hypothesis readily explains the selective action of the Roentgen treatment and shows why effective dosage is difficult, since it must be strong enough to injure pathological cells and yet not too strong for normal tissues. Fortunately in practice the reaction of the skin forms a valuable clinical guide.

The clinically demonstrated physiological action of this agent, whatever it really may be, is at least unique, since it possesses the power of inhibiting the growth of malignant cells, possibly of destroying them. No other agent has ever demonstrated such power over the morbidity or the cellular energy of growth of malignant tumors. No other agent has possessed the selective power to destroy malignant cells without injuring the normal surrounding structures. The only effective method of treatment has been the removal of the diseased tissue with the surrounding normal structure or their total destruction *in situ* by cauterants. The great difference between this and all other agents employed in treating malignant disease is that it differentiates between pathological and normal tissues, retarding and preventing the development of pathological cellular activity, while it does not harm or hinder the growth of normal cells.

The latest views regarding malignant growths attribute their malignant character to an inherent and unexplained increase in the energy of growth of their cells, which experimental studies (Loeb) have shown is in marked contrast to that of the cells of normal tissues or of benign tumors. This energy varies in intensity in different tumors, but is constant for each tumor even through a long series of transplantations. Further, it has apparently been demonstrated that the formation of metastases is caused and can occur only where there is a primary increase in the energy of growth of the tumor cells.

These pathological facts harmonize with the physiological action attributed to the Roentgen energy. In their light the rationale and value of preparatory or tentative Roentgen treatment before operation and of postoperative treatment can be seen. The peculiar primary energy of growth of malignant cells is reduced by the inhibitory action of the rays. The growth not only of the original tumor but also of metastases and wandering malignant cells is checked.

It is generally conceded that an irritating or exciting cause is an essential to the development of malignant growths, and that often incomplete operations act as exciting causes and increase the morbidity of the growth. Preparatory and postoperative treatment are, therefore, valuable, because they inhibit the energy of malignant cells, if they do not destroy them, and render metastasis less liable, while the supplementary treatment destroys any remaining cells or inhibits their growth.

Can it be possible that in these pathological findings and physiological action an explanation is found for the production of malignant growths in the hands of Roentgen operators? Such cases have, unfortunately, been reported, and in them the disease originated simultaneously in both hands. Possibly the continued though feeble stimulation of normal cells resulted in a permanent increase in their energy of growth, whereby they took on the characteristics of malignant cells and acquired the power to grow and multiply at the expense of surrounding normal tissues and to form metastases.

An analogous process as diametrically opposed as is the destruction and production of malignant disease is seen in the clinical effects in hirsuties and alopecia areata, where overstimulation destroys the hair and mild stimulation causes an increased metabolism in the hair follicles and a renewed growth of hair.

But what are the difficulties that underly the accurate application and dosage of this agent? Why have the results been so varied and antagonistic? Why should they be better? And what is essential to its efficient and successful application?

The chief difficulties that underly the accurate application and dosage of this therapeutic agent lie in its method of production and the variation in its qualities, that must be recognized and applied accurately to be effective. The complicated physical apparatus employed in its production must be thoroughly understood, and special knowledge and technique acquired to produce and recognize the various qualities of this therapeutic force. In addition, clinical experience and knowledge are essential to an appreciation of their effects and the adaptation of the particular quality of energy to the lesion which it most readily affects.

Improvements in apparatus for the production and measurement of the amount of energy that is developed make these difficulties less and the acquirement of technique easier; but there will always

remain the problems common to the employment of all remedies in clinical work. As has been aptly said of this agent, it must be applied in the proper quality, dose, and frequency. The most specific drugs known, quinine and mercury, cannot be successfully applied without accurate diagnosis and the help of clinical experience in dosage.

With the necessity of developing special technique to produce this therapeutic agent accurately, and again clinical technique and experience in employing it, is it to be wondered at that results have been often disheartening to those who have expected any quality and any dose to accomplish results? When, in addition, it is remembered that the majority of cases treated, especially of malignant disease, have been hopelessly inoperable, is it not a proof of the inherent value of this agent that any successes have been recorded?

The recognition of the necessity of adapting the quality and dose of this agent to each individual lesion and patient, and a clearer knowledge of which quality and technique is applicable to each group of cases, afford the prospect of greater developments and of more frequent successes in the future.

Efficient Roentgen therapy is not the result of chance, nor can it be purchased with the apparatus. The wonderful results that have been produced, though they may not be always repeated, were not due to good fortune or accident. They have followed diligent, untiring study of the phenomena observed in the Roentgen tube and their clinical effects upon disease. The necessity for special education of the clinician who would apply this agent effectively is self-evident. The knowledge of how to produce the required quality of Roentgen discharge must be combined with the knowledge of when to apply it, and clinical experience must be capable of measuring the exact dose. No therapeutic agent can be effective without the fulfilment of these requirements, and there is little wonder that without them more failures than successes have been recorded.

Such a powerful therapeutic agent is dangerous or useless in the hands of anyone less well qualified. It is dangerous if he employ it in a dose capable of doing good and is incapable of accurately observing its effects; and it is useless and even harmful if inexperience makes him timid in its employment, or ignorance leads him to employ a dose that will stimulate instead of injure pathological tissues.

But are not the poor results in deep-seated disease due to the limitations of this therapeutic agent, or why is it that so many can produce effects upon superficial lesions and cannot influence the deep-seated? There was a similar condition in the field of Roentgen diagnosis; at first lesions of bone in fingers, hands, and toes only could be diagnosed; as technique improved all parts of the body have been penetrated and the various tissues have finally yielded

to a differentiation in their shadows. Those who can produce therapeutic effects on superficial lesions only will often be found as defective in making diagnoses of deep-seated lesions. Any technique and any even inefficient apparatus will effect superficial lesions just as they will make pictures, and some diagnoses in the bones of the hand and arm. The progress which has marked the development of improved technique in Roentgen diagnosis will find its counterpart in Roentgen therapeutics. The effects of varying qualities of Roentgen rays in therapeutics is now being studied as were their effects upon diagnosis. There is a more accurate application and greater efficiency already manifest. Three years ago it took twenty-eight treatments to cure an epithelioma of the eyelid which has remained cured since. A similar growth was removed apparently as effectively this winter in fourteen treatments. The result was due to an improved technique and an adaptation of the fitting quality of ray to the disease. The qualities peculiarly adapted to each variety and situation of disease are being gradually determined; many of them are already known. Advances and better results have followed and will follow each improvement in technique.

The means for measuring the amount of energy flowing through the tube have been improved, so that it is possible to compare the technique of different operators more accurately and to determine in what the effective treatment for each lesion consists.

When and how should this therapeutic agent be applied to make it most effective and of the greatest benefit to the patient?

In malignant disease it should undoubtedly be employed in conjunction with operation, not as a substitute for it, but as a component part of the treatment. Up to the advent of the Roentgen method of treatment early radical surgical removal of so much of the diseased tissues as was possible was the most efficient treatment known. That, however, cannot be as truly said now. The efficiency of the Roentgen treatment has been demonstrated to be great; it is capable of modifying the malignant nature of the cells, retarding their growth if not totally destroying them. Surgery cannot, therefore, be said to be the most efficient treatment unless it is combined with thorough postoperative Roentgen treatment.

Surgical operation and Roentgen treatment are not antagonistic, but mutually helpful and supplementary, and the patient's greatest good can be served only when they are combined. The time for applying the Roentgen rays should be either immediately after operation or just before, as a short preparatory treatment to reduce the energy of growth of the cells and destroy the outlying cells, and then again after operation.

The patient has the best chance of permanent relief from malignant disease who is brought early for operation, and that operation followed immediately by a thorough course of Roentgen treatment

The treatment must be as severe as the patient can stand, both locally and through the systemic effects, if it is to be of value.

A marked dermatitis or tanning of the skin must be produced or sufficient severity in the dose has not been employed. Auto-intoxication from the absorption of the dissolved malignant cells must be expected if efficient dosage has been employed where there are large growths. In cases where treatment is efficient this auto-intoxication will manifest itself in rheumatic pains in muscles and joints, disturbances of the stomach and liver, and a general feeling of malaise. The detection of these symptoms is an indication for the interruption of treatment for one or two weeks, as its continuance would put the patient in bed. It is, however, an indication of effective treatment. Such symptoms can, however, be produced only where there are large masses of tissue of low vitality that can be affected, as in massive recurrences or inoperable growths. It is to obviate this absorption and to hasten treatment that palliative operations can be employed in practically hopeless cases before the Roentgen rays are used. The inhibitory action, together with the ability to relieve pain where nerve trunks are not involved in the disease, makes the Roentgen rays the most potent palliative remedy known in the late stages of malignant disease.

A CASE OF UNIVERSAL CONGENITAL ATRICHIA.

By Augustus A. Eshner, M.D.,
of Philadelphia.

The unique case I am about to report is one of total absence of hair from all parts of the body, existing, so far as could be learned, from birth. If the history, as given by the patient, is correct, the condition cannot, strictly speaking, be regarded as one of alopecia or baldness, that is, loss of hair, but, rather, as one of atrichia, namely, failure on the part of the hair to develop. The universal distribution of the disorder is not the least remarkable feature. In addition, the nails exhibited significant changes, and there was present, besides, the rare anomaly of retinitis albicans.

The patient was a house-painter, aged sixty-four years, who was admitted to the Philadelphia General Hospital on January 2, 1904, complaining of pains throughout the body, especially in the lumbo-sacral region. He presented slight swelling of both lower extremities and drooping of the right upper eyelid, with inability to elevate the lid properly. The action of his heart was arrhythmic, but the sounds were clear. The especially noteworthy feature about the patient was the total absence of hair from all parts of the body, a

condition that, according to the patient's repeated statement, had existed from birth. The skin itself was soft, smooth, and unctuous. The man related, further, that he perspired but little. The nails of the fingers and toes were only about one-half the normal length, and their distal extremities were irregular and longitudinally rugous. The patient stated that they had presented this appearance from birth and that he never trimmed them. He had only three teeth in the lower jaw and none in the upper; the others had gradually fallen out in the progress of time. The urine was free from albumin and sugar.

Dr. C. A. Oliver kindly examined the eyes and confirmed the absence of lashes. In addition, he found the condition of retinitis albicans, characterized by the presence of numerous mosaics and areas of whitish masses, with exposure of the underlying sclerotic. So far as the patient knew, no other member of his family exhibited a like absence of hair or alteration in the nails. He gave a personal history of gonorrhœa and of lead poisoning; there was some doubt as to syphilis.

The accompanying illustrations show the absence of hair from the head and the face, including eyebrows and eyelashes. Negatives obtained from exposure of the entire body, and of the hands separately to show the finger-nails, proved unsuccessful, and the patient declined to be exposed again.

Whether the absence of hair in this case had really existed from birth, as the patient maintained, cannot, of course, be established with certainty, as we have only his unconfirmed statement to this effect. In any event, the condition, whether one of generalized loss of hair or one of generalized failure on the part of the hair to develop, is exceedingly rare. I have been able to find records of only a few similar cases, and to some of these the references are exceedingly brief, and in some instances indefinite.

F. G. Danz[1] refers briefly to two grown brothers who had always presented entire deficiency of both teeth and hair.

Steimnig[2] reports the cases of a boy, aged three and a half years, and his sister, aged one year, whose bodies at birth were devoid of hair and covered only with vernix caseosa. Nowhere on the head was there even a trace of lanugo, and eyebrows and eyelashes alike were absent. The nails of the fingers and toes were only indicated, appearing as shrunken or shrivelled points. The children sweated freely, especially about the head. The parents were healthy and had each a strong growth of hair.

John F. South[3] refers to the case of a man, aged twenty years,

[1] Stark's Archiv f. die Geburtshülfe, Frauenzimmer- und Neugebohrner Kinderkrankheiten, Bd. iv., 3. Stück, Jena, 1792, p. 684.

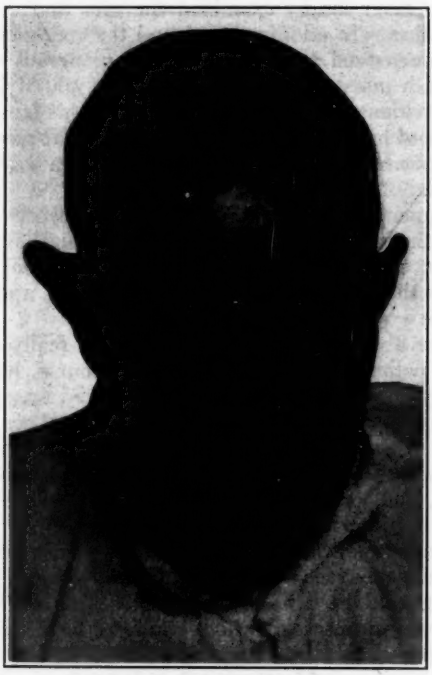
[2] Notizen aus dem Gebiete der Natur- und Heilkunde, Bd. xxvi., No. 4, November, 1829, No. 554, p. 50.

[3] Translation of Adolph Wilhelm Otto's Compendium of Human and Comparative Pathological Anatomy, London, 1831, p. 120.

who had no hair on his head, eyebrows, eyelids, or chin, and was said to have none on the pubes.

Burkard Eble[1] cites Laurent Heister,[2] as speaking of two individuals that had no hair on the entire body; and as having seen a man, aged about forty years, and otherwise well, who, some ten years previously, had, without recognizable disease, lost all of his hair, including the eyebrows and eyelashes, so that no trace could be discovered; and also a woman, who, without antecedent

FIG. 1.



Universal congenital atrichia; front view.

disease, had lost all of her hair. He refers to observations of individuals without hair or teeth recorded in the *Transactions of a Society of London* for 1800, and the *Salzburger medicinisch-chirurgische Zeitung* for 1801, vol. i. p. 250; quotes Schenk as authority for the statement that King Louis of Hungary was born without

[1] Die Lehre von den Haaren in der gesammten organischen Natur, Vienna, 1831, Bd. ii., p. 244.

[2] Medicinische, chirurgische und anatomische Wahrnehmungen, Rostock, 1753.

hair; cites the observation by Dr. Wells[1] of similar complete absence of hair, by H. F. Delius of an individual without even the smallest hair upon the body, and by Augustin[2] of a man on whose body no trace of hair could be observed; and speaks of a similar case mentioned by Leveling.[3]

M. Schede[4] has reported the cases of a brother and sister, aged thirteen years and six months respectively, who were hairless at birth and continued to be so up to the time of observation, when no

FIG. 2.

Universal congenital atrichia; back view.

trace of hair could be discovered on any part of the body. The parents and two other children intermediate in age were healthy and exhibited a full growth of hair. A piece of scalp was excised from the head of the elder of the children in question, and well-

[1] Transactions of a Society for the Improvement of Medical and Surgical Knowledge, 1800, vol. ii.

[2] Asklepieion, March, 1812, Heft 3.

[3] Haller's Grundriss der Physiologie, Erlangen, 1795, Bd. viii., p. 1. Theil, note 325, p. 384.

[4] Archiv f. klinische Chirurgie, 1872, Bd. xiv., p. 158.

developed, sebaceous glands were found opening on the surface, while at their base were sebaceous cells having distinct nuclei and fragmented contents. Nearer the orifice the cells formed irregular sebaceous masses. Close by were a large number of small and large atheromas separated from the glands by connective tissue, evidently developed from glandular tubes. All of the latter, however, were not distended or degenerated. In places they were short and straight or slightly convoluted and not always provided with a lumen. They were filled with cells arranged in two layers, an outer, consisting of long, narrow cylinders, with rod-shaped nuclei, and an inner, consisting of small, round, or polygonal cells. These were looked upon as rudimentary hair follicles. The sweat glands were not normally developed. The erector muscles of the hair were well developed, but there was no sign of hair.

J. Higham Bell[1] refers to an aboriginal black man and woman, brother and sister, said to be entirely devoid of hair, a sister of whom, similarly hairless, had died a few years before, and who had, when young, been brought to a station in Australia in the same hairless state. They were thought to belong to a tribe of hairless Australian blacks. Photographs represented a well-formed and well-developed man and woman of middle age, without a particle of hair visible on their heads and bodies, and none was found on most careful inspection. The peculiarity is especially striking, as the Australian aborigines are naturally a hairy race.

In a paper on so-called dog-men, Alexander Brandt[2] refers to a Russian peasant with absolute congenital atrichia mentioned by one of his students. He points out that the fetal lanugo may either fall out and not be replaced by permanent hair or be excessive and persistent, and give rise to the condition of abnormal hairiness.

An additional number of cases have been reported in which there has been temporary or transitory absence or loss of hair, generalized or local, natal or postnatal, often in conjunction with abnormal conditions of the nails and the teeth, sometimes with deficiency or total absence of perspiration; in one instance with absence of the secretion of tears, and in one with absence of the mammary glands; frequently there was a familial distribution.

Rayer[3] reports the case of a man whose cranium appeared entirely naked, although, on close examination, a quantity of fine, silky, white down was found present. There were also a few black specks on the temples representing stumps of hairs that had been shaved off. The eyebrows were indicated by fine short hairs. The edges of the lids were without lashes, but the bulb of each of these was indicated by a small, whitish point. The beard was thin and weak. There were

[1] British Medical Journal, January 29, 1881, p. 177.

[2] Biologisches Centralblatt, March 1, 1897, Bd. xvii., p. 161.

[3] Theoretical and Practical Treatise on Diseases of the Skin, second edition, translated by R. Willis, London, 1835, p. 1049.

a few straggling hairs on the breast and pubic region, and scarcely any in the axillæ. Hair was more abundant on the inner aspect of the legs. The father of the patient presented a similar defect.

John Thurnam[1] has reported two cases in cousins exhibiting imperfect development of skin, hair, and teeth. One occurred in a man, who died at the age of fifty-eight years, and was, during life, almost completely without hair. He had only four teeth. There was a small amount of fine hair on the head and face, in the axillæ, and on the pubes. The eyebrows were absent, and the eyelashes were defective. The skin generally was fine, thin, and delicate, and its surface was often dry and harsh, without traces of sensible perspiration and any trace of unctuosity. Sudorifics made the skin soft and relaxed, but had no effect upon its state of moisture. A trace of fluid appeared upon the surface in the last week or ten days of life. There was, further, an absence of tears. After death the skin was found in a condition of atrophy and atony, and the sudoriparous apparatus defective. The second case was in a maternal cousin who exhibited almost precisely the same peculiarity, the head being equally bald, the skin similarly delicate in structure, the sensible perspiration and tears likewise absent, and the teeth also deficient. The maternal grandmother exhibited extreme delicacy of the skin, with a limited amount of sensible perspiration.

In discussing Thurnam's case, Williams[2] referred to the case of a girl, aged fifteen years, described as being devoid of hair and teeth, but found to be not entirely so. The hair was fine, scanty, and white, although there was scarcely any forming the brows and lashes. The patient had three or four projections resembling teeth, and these were carious. She had never perspired, and menstruation had not yet set in.

William Sedgwick[3] cites the case, reported by T. H. Burgess, of a boy, aged eight years, who was without a vestige of hair on the scalp, devoid of eyebrows and eyelashes, and without hair on any part of the body. He had the usual supply of hair until the age of four years, when it began to fall out gradually, until it had disappeared entirely. The brother of this boy, two years younger, had begun to lose his hair in detached places. His sisters, younger and older, had a good supply of hair, as had also the parents.

H. MacNaughton Jones and Ringrose Atkins[4] describe the microscopic appearances in a case of so-called congenital alopecia. The patient was a boy, under treatment for his eyes, who had no recollection of ever having had any hair on his scalp, although he had heard during youth of the presence of some downy hairs. The face was the only part of the body on which there was any appearance of

[1] Medico-Chirurgical Transactions, 1848, vol. xxxi., p. 71.
[2] London Medical Gazette, 1848, vol. vi., p. 336.
[3] British and Foreign Medico-Chirurgical Review, 1863, vol. xxxi.
[4] Dublin Journal of Medical Science, 1875, vol. lx., p. 200.

hair, and here there was a fine down. The finger-nails were aborted and badly formed, with ridges and furrows. The teeth were irregular, far apart, and marked by transverse ridges and discoloration. Histological examination of sections of the scalp disclosed the presence of irregular tubules, crossed by fine trabeculæ, and containing granular material, and also in places an aggregation of circular and ovoid apertures deeply lined with small and compressed epidermic cells. These apertures appeared to be transverse sections of tubular cylinders, and these were thought to represent hair follicles altered in situation, position, and structure. Here and there was an aborted hair follicle, forming a shallow pit in the epidermic layer and lined by a layer of similar cells, the open extremity looking downward.

J. B. Luce[1] reports the case of a girl, aged eight and one-half years, who was said to have been entirely bald at birth. In the sixth month a number of small elevations appeared on the scalp, and the first hairs were observed at the age of six years. At eight and one-half years numerous lanugo hairs were present, and also a little blackish-gray hair. The scalp was the seat of conical nodules, the skin over which presented a normal color, with a central dark point. Introduction of a needle into such a nodule permitted the escape of a rolled-up hair.

Paul Michelson[2] cites the case of a girl, aged two years, otherwise normal, whose head was sparsely covered with lanugo.

S. H. Guilford,[3] of Philadelphia, describes the case of a man, aged forty-eight years, edentulous from birth, who exhibited absence of the sense of smell, almost complete absence of the sense of taste, absence of sweating and of hair from the trunk, with an excess of hair on the face, in the axillæ, and in the pubic region, while the head was covered with soft down. His maternal grandmother had never had hair or teeth, while his mother was normal in these respects. A brother of the latter also was without teeth and hair. Of her twenty-one children, eighteen were living, but the patient was the only one without teeth, although in some certain teeth had failed to make their appearance. The patient himself had eight children, several of whom were in some degree edentulous, although otherwise normal.

Jonathan Hutchinson[4] has reported a case of congenital absence of the hair and the mammary glands in a boy aged three and one-half years. The head, however, was covered by a quantity of down, and the teeth were normal. The nails of the fingers and toes were thin and curved backward, presenting a depression in the middle. The nipples were replaced by cicatricial patches, but there was no

[1] Thèse de Paris, 1879, No. 579; Schmidt's Jahrbücher, Bd. cxci., Heft 1, p. 31.

[2] Handbuch der Hautkrankheiten in Ziemssen's Handbuch der speciellen Pathologie und Therapie, Leipsig, 1883, p. 107.

[3] Wiener medizinische Wochenschrift, 1883, No. 37, p. 1116.

[4] Medico-Chirurgical Transactions, 1886, vol. vii. p. 473.

trace of the mammary glands. The mother had, from the age of six years to the time of the report, suffered a gradual loss of hair from alopecia areata. At first the scalp became bald and smooth; subsequently the eyebrows and eyelashes fell out. The latter grew again, and a few tufts of hair appeared on the scalp.

This case has been more recently described by Hastings Gilford[1] as one of progeria, or premature senility. The patient was at that time fifteen and one-half years old. On close inspection a feeble growth of colorless hair was found on the back of the hands and wrists and on the head. The teeth were of good size and in fair condition. In another case, reported by Gilford,[2] the patient was a young man, aged eighteen years, whose eyebrows and eyelids at first appeared devoid of hair, but on close inspection a few scattered and downy hairs were discovered in both situations. A small number were detected also on the backs of the hands and wrists. None, however, could be found upon any other part of the body. The nails of the fingers and toes were short, ill-shaped, and membranous.

Paul de Molènes[3] reports the case of a girl, aged four and one-half years, who at birth presented an almost imperceptible down on the scalp, scarcely developed eyelashes, and normal nails, with an absence of eyebrows. At the age of five months the eyelashes fell out, and at the age of sixteen months the entire hairy system was deficient. On the scalp no down was visible, even with the aid of a lens, although numerous orifices were present, indicative of the integrity of the pilosebaceous organs. A fine white down was discernible on the lower lids with the aid of a lens. The nails and teeth were normal. Under treatment slow and gradual growth of hairs took place in various situations. Hairs and follicles were found normal on microscopic examination. The mother of the patient had been treated for extensive alopecia areata at the age of nineteen years, and a brother at the age of six years.

Molènes states that the condition of atrichia is exceedingly rare and almost never absolute. Generally a fine down is present, indicating the existence of hair follicles. Usually, also, the anomaly is partial. It may persist for several months after birth, and even up to the second year. Few examples are known in which it persisted throughout life. Molènes considered it a perversion of function or an arrest of development affecting the organ giving rise to the hair.

Two cases of congenital familial alopecia were exhibited at the meeting of the Société Française de Dermatologie et de Syphilographie, held November 10, 1892. The condition could not have been complete, as the statement is made that the hair was the seat of a garland-like malformation.[4]

[1] Practitioner, August, 1904.
[2] Ibid.
[3] Annales de dermatologie et de syphilographie, 1890, tome i., p. 548.
[4] Monatshefte f. praktische Dermatologie, 1892, Bd. ii., p. 618.

Aubry[1] reports a case of congenital alopecia in a young man, aged sixteen years, in which the loss of hair followed, in a general way, the lines of the cranial sutures, the remainder of the head being covered with a dense layer of black hair.

The condition was thought to be due to atrophy of the skin from stretching, in consequence of hydrocephalus. The teeth were normal.

At a meeting of the New York Dermatological Society, held November 27, 1894, J. A. Fordyce[2] presented a girl, aged four years, with complete alopecia. A few hairs were noticed at birth, but these soon fell out. A few scattered eyelashes were present. There was no family history of alopecia.

P. S. Abraham[3] reports three cases of congenital alopecia in a mother and her two children. The former, aged thirty-three years, exhibited complete absence of hair from the eyebrows, eyelids, arms, legs, trunk, with a scanty supply on the scalp, pubes, and axillæ. At birth there was a small fluff on the head, but this soon fell out. The absence of hair remained complete until the age of eighteen years, when hair gradually made its appearance on the scalp, in the axillæ, and on the pubes. Of the children, one, aged five years, was in a state of almost complete alopecia. There was a little down on the head at birth, but this fell out at the age of three months. The second child, aged fifteen months, likewise appeared to be completely without hair. It had black hair at birth, but this fell out at the age of three months.

C. Nicolle and A. Halipré[4] have described a condition of deficiency of the hair, with trophic changes in the nails, occurring in thirty-six members of one family during six generations. He reports at some length the case of a young man, aged eighteen years, deficient mentally, whose eyebrows were scanty and unpigmented, the lashes white, the hair on the scalp thin and in places lighter in color than elsewhere. There was no hair on the anterior aspect of the chest, only a few short, white hairs in the axillæ, no mammary hair, and a moderate amount of long brown hair on the pubes. The teeth were normal. The terminal phalanx of each finger was larger than normal, œdematous and red, while the nails were in varying degree hypertrophied, longer and thicker than normal, rugous and scaly, with a tendency to become elevated and incurvated at the extremity. The majority exhibited exaggeration of the longitudinal striation, and some were striated transversely. They were extremely friable, some being cracked in the middle and others longitudinally. Some were separated and tended to become detached. The free border was, in general, black, the rest of the nail grayish-yellow. The

[1] Annales de dermatologie et de syphilographie, 1893, 3. ser., tome iv., p. 899.
[2] Journal of Cutaneous and Genito-Urinary Diseases, vol. xiii., p. 120.
[3] British Journal of Dermatology, 1895, vol. vii., p. 162.
[4] Annales de dermatologie et de syphilographie, 1895, 3. ser., tome vi., p. 804.

extremities of most of the fingers about and beneath the nails were the seat of ulceration.

A case of similar character is reported by Charles J. White.[1] This occurred in a young man, aged nineteen years, who presented an abundant uniform growth of pale, short, downy hair on the scalp, with an absence of hairs from the cheeks, the chin, and the axillæ. The eyelids and eyebrows were normal, the hair on the upper lip of the lanugo type, and a few hairs were present on the pubes. From the age of nine years, following an injury to two of the fingers of one hand, the bed of the nails of all of the fingers and toes was from time to time the seat of ulceration, the nails becoming thickened and broken and discolored at the extremities. Staphylococcus pyogenes aureus and staphylococcus pyogenes were obtained on culture. The hairs presented a normal appearance. The great-grandfather of the patient had had little hair, and the nails of his fingers and toes were faulty in development. The second generation escaped. The third generation comprised eight children, six sons and two daughters, of whom one son and two daughters were affected—the mother and two uncles of the patient. The former had always had but little hair, and the finger-nails had for years been thickened and abnormal. The uncles had had normal hair, but the nails were bad. One of the uncles married and had two children, a son of nine years and a daughter of four years. The latter was born without hair or nails, and these failed to develop. The patient had one sister, two step-brothers, and one step-sister. The sister had a thick, downy head of hair, the individual hairs being blonde and short.

Paul Ziegler[2] has reported the case of a girl, aged seventeen years, who was hairless at birth, though otherwise normal. She was the youngest of eleven children, all of whom but her presented no abnormality. There was no family history of aberrant growth of hair. From the age of thirteen years a small bunch of black hair made its appearance every four weeks at the occipital protuberance, disappearing in the course of four days with the cessation of menstruation. From the same period, also, there had been a small amount of down on the cheeks. A year previously a few hairs of normal appearance had developed on the eyebrows and eyelids. Recently the patient had noticed fine hair on the forearms. The teeth and nails exhibited no peculiarity. The arms, shoulders, and thighs were the seat of lichen pilaris. On microscopic examination of a bit of skin removed from the scalp, the epithelium and the sebaceous glands were found normally developed, with numerous papillæ, but with an absence of hairs. Near the sebaceous glands, generally at their bases, at a distance from the surface epithelium, were isolated convoluted epithelial tubes, with a large, generally circular, lumen and not communicating with the surface. These

[1] Journal of Cutaneous and Genito-Urinary Diseases, 1896, vol. xiv., p. 220.
[2] Archiv f. Dermatologie und Syphilis, 1897, Bd. xxxix., p. 213.

were lined with from four to six layers of cells, and they were surrounded by connective tissue, near which were unstriped muscular fibres—erectors of the hairs. Similar fibres were present also in the vicinity of the sweat glands, which themselves were normal in every respect. The epithelial tubes were thought to represent the remains of the external sheath of the root of the hair.

Ziegler ascribes the failure of the hair to develop to local alterations in the external sheaths of the root of the hair below the level of the excretory duct of the sebaceous gland. He thinks excessive production of the cells of the sebaceous duct may cause occlusion of the lower portion of the sheath of the root of the hair, resulting in a constriction off of the lowermost portion of the sheath before the papilla has formed. In some instances he believes there may be delayed growth of otherwise normal hair.

Felix Pinkus[1] has reported the case of a boy, aged eight years, who at birth had a growth of hair on the head, especially on the posterior portion. The forehead was bald, but it was not known if there was any hair on the body. At the age of a few months a deposit of crusts formed on the vertex, and this was followed by loss of the hair. The child was quite bald at the age of nine months, and thereafter only a few hairs grew. The body was at this time almost hairless. A year later there were only a few short hairs at the root of the penis, and only a few on the back from the neck to the buttocks. There was scarcely any hair on the arms and on the legs. The face was the seat of abundant lanugo, with a few stray hairs on the forehead and the cheeks, while the lobule of the ear and the tragus were well supplied with hair. The eyelashes were well developed, while the eyebrows were deficient. On the head the hair was scanty, irregularly distributed and short, though requiring cutting from time to time. The hairs were thin, dark, and twisted. The nails were thin, flat, unduly white, and friable at the edges, and their surface was covered with bands converging to the tips of the fingers. The teeth were in process of change, and some had been extracted. The second premolars were wanting, as well as the left lower canine and both outer upper incisors.

Pinkus employs the term hypotrichosis to designate the condition present in this case, contending that the pathological state does not consist in a loss of hair, but rather in deficient growth, the failure occurring at the time when the lanugo or deciduous hair ordinarily is replaced by the permanent hair. The affection would thus be a developmental defect and not properly a disease.

H. Radcliffe Crocker[2] refers to the case of a girl, aged four years, born without hair or nails. The latter began to grow abnormally within a week, the former not for three years. In addition, there was atrophy of the skin generally.

[1] Archiv f. Dermatologie und Syphilis, 1899, Bd. l., p. 347.
[2] Diseases of the Skin. P. Blakiston's Son & Co., 1903.

PRIMARY CARCINOMA OF THE OMENTUM WITH PERITONITIS CARCINOMATOSA.[1]

By Arnold Sturmdorf, M.D.,
of New York.

While it is not unusual to find the omentum involved in malignant disease, either by contiguity or metastasis from adjacent or remote organs, it is rare to find it the site of a primary malignant growth.

Such primary peritoneal cancers have been the subject of much investigation and controversy and there still exists much confusion in terminology as well as in fundamental conceptions as to histogenesis and development.

For obvious reasons the older publications on this subject cannot be utilized in the elucidation of such obscurities from a modern point of view.

Thus, it was formerly accepted that the histological characteristics of primary peritoneal cancers were typically *carcinomatous*, while within the last decades this view has undergone considerable modification.

It became apparent that the primary development of an epithelial growth, in parts in which epithelium does not normally exist as such, is incompatible with advanced conceptions of histogenic and biologic processes.

Consequently the term *carcinoma* applied to such primary peritoneal neoplasms must prove a misnomer and, as a result, either the *primary* nature or the *carcinomatous* type of the cases reported was questioned, so that in 1872 Waldeyer wrote:

"Whereas, before the appearance of Thiersch's work on this subject, cases of primary carcinoma of the peritoneum were described by a number of observers, no reports of such cases have appeared within the last six years."

The reported cases alluded to in this quotation were discarded by Orth as unauthentic and lacking in definite characterizing detail.

Notwithstanding these theoretical contentions, we find, three years later (in 1875), the report of a case of primary carcinoma of the peritoneum published by Quincke; while again, in 1890, the Committee on Morbid Growths of the Pathological Society of London examined and described as carcinoma, a primary tumor of the omentum with numerous metastatic growths in the abdominal cavity shown by West.

Whether these two authoritative cases really demonstrate the heterologous development of primary carcinoma, must remain an open question until definite knowledge is gained concerning the

[1] Presented before the Surgical Section of the New York Academy of Medicine November 4, 1904, with presentation of specimen and microscopic demonstration.

characterizing elements of the group of tumors embraced by the generic term *endothelioma*.

These latter growths, said to arise from the endothelial layer of lymph- and bloodvessels, may so closely simulate carcinoma in microscopic appearance that their endothelial type can be established only by serial sections, locating their origin in or about the lymphatics and bloodvessels.

The exact histological position, however, of such endotheliomata is still in dispute.

They have been regarded as infectious, and Leyden succeeded in isolating a peculiar form of rhizopod from the ascitic fluid of a case of primary malignant peritonitis.

This finding has been corroborated in a number of cases by Nothnagel, Lauenstein, and Schaudinn.

The last proposed the term Leydenia gemmipara for these organisms, although their etiological relationship to peritoneal cancer is not at the present time generally accepted.

Delafield and Prudden describe endothelioma as "a group of tumors which on the one hand are closely related to the sarcomata in genesis and in some cases in appearance, while on the other hand some of them so closely resemble some forms of carcinoma as to be difficult of distinction from them."

This bizarre nature of the histological criteria in these tumors explains the contradiction as to prevalence and frequency found among authoritative reports.

Thus, Glockner, who analyzed the literature on the subject to 1895, states that "primary peritoneal endothelioma is by no means rare," while Nothnagel mentions but two personally observed cases in which the peritoneum was primarily and exclusively involved.

It is possible that the direction of modern etiological research, in relation to malignant growths in general, may ultimately point the way to an elucidation of some of the obscurities thus briefly outlined; in the present instance, however, it is only possible to adhere to the prevalent caption, carcinoma, leaving it to more advanced doctrines to relegate the cases into their proper category.

The history of our case is as follows:

Mrs. E. C., aged sixty-two years, married thirty years, widow eight years; father died of Bright's disease; mother of pulmonary tuberculosis. Patient was normally delivered of three children, the last twenty-six years ago, all of whom died of tuberculous disease. Menstruation began at fourteen and ceased permanently twenty-five years ago. No menstrual or climacteric abnormalities.

Barring a traumatism which resulted in the acute destruction of her left breast twenty-six years ago, her previous history presents no data bearing on her present condition.

For four months an increasing tenderness, with recurring attacks of pain, accompanied the gradual development of a hard mass,

occupying the middle of the right half of the abdomen. The mass grew rapidly, while pain, abdominal distention, dyspnœa, with loss of flesh and strength, developed to a distressing degree. When first examined, in August of this year, the patient presented the following status: A pale, yet fairly nourished woman of medium stature, with normal pulse and temperature; slight dyspnœa. The heart presents evidences of a compensated mitral insufficiency. Lungs normal; abdomen symmetrically enlarged and very tense.

General abdominal tenderness, most marked in the right half, which contains the major part of a hard, quadrilateral mass, the upper border of which can be defined as a nodular ridge extending across the epigastrium from points continuous with the mammillary lines. The lateral borders of this mass converge (the left more than the right) toward its lower pole, which seems to project free into the pelvic cavity. The mass is very hard and presents a rough, irregularly nodular surface.

It is uninfluenced by respiration, is slightly mobile from side to side, and to a less degree in an upward direction. Its upper border is fixed, while its lower free pole could be subjected to ballottement in the excessive amount of ascitic accumulation. Percussion and colonic inflation indicated the presence of intestine between the liver and the upper border of the growth. Spleen not demonstrable. Genito-urinary organs intact and free from involvement. Marked œdema of ankles. Urine normal. Blood shows 60 per cent. hæmoglobin, no corpuscular changes—quantitative or qualitative.

On September 14th, through a four-inch supraumbilical incision, which gave vent to a large quantity of serosanguineous fluid, a mass of infiltrated omentum was removed. Its removal offered no unusual difficulties. The parietal peritoneum was found studded with countless papillary excrescences, presenting hard, flattened nodules, varying in size from miliary dots to a split pea, very hard, yellowish-white in color, and in many instances showing a tendency to umbilication. Each papilla presented a small area of inflammatory margin around its base. Aside of this finding, the most painstaking search throughout the abdominal contents failed to reveal the presence of similar neoplastic formation as a possible primary or secondary focus of the disease.

Although the cancerous nature of the growth was evident at the time of its extirpation, yet the clinical resemblance to some forms of peritoneal tuberculosis, more especially in view of the tuberculous family history, made a positive differentiation impossible until Dr. Schnaper kindly furnished the microscopic evidence of its carcinomatous character, which I beg to submit for your inspection. He also furnished the following report:

"The tumor upon microscopic examination presents the characteristic picture of an alveolar carcinoma. The epithelial cells are varied in shapes. Nucleus stains well. The epithelial masses are

varied in size and shape; the cells fill the alveoli and are in close relationship to one another. The Van Gieson stain failed to reveal any stroma between the cells. The stroma is made up of firm connective tissue and shows infiltration of fat, and in places there is a round-cell infiltration."

Whether true carcinoma or endothelioma, and if carcinoma, whether primary or secondary, the primary focus having thus far eluded our search, will probably remain an open question for the present. The patient made an uninterrupted recovery from the operation. It is now seven weeks since, and she presents no functional disturbance that might point to the possible involvement of a particular organ and is apparently enjoying the results of a symptomatic cure.

While the case thus detailed presents many points for profitable discussion, both practical and theoretical, there is but one phase of which I would speak in closing, and that is the complicating carcinomatous peritonitis mentioned in the title.

The multiple papillary excrescences found studding the parietal peritoneum cannot be interpreted in this case as merely passive metastatic deposits, but represent a type of malignant subacute efflorescence, clinically similar to that which is recognized as occurring in tuberculous and other infectious processes, and productive of profuse serous exudation. As a matter of fact, such forms of primary acute miliary carcinosis of the peritoneum are described by Bauer in *Ziemssen's Cyclopædia* and by Nothnagel, the miliary excrescences resembling those described above in every detail. In no other way can we explain such enormous ascitic accumulation in so short a period in the absence of circulatory obstruction.

A complete bibliography will be found in *Ziemssen's Cyclopædia*, vol. viii. p. 337; Nothnagel, *Specielle Pathologie und Therapie*, article "Geschwülste d. Peritoneum."

HYPERTROPHY AND STENOSIS OF THE PYLORUS IN INFANTS.*

By F. L. Wachenheim, M.D.,
of New York.

The following addition to the sixty-odd hitherto recorded cases of hypertrophy and stenosis of the pylorus in infants has come under my observation:

Thomas M., aged five weeks, was seen by me for the first time on September 29, 1904. His mother stated that uncontrollable

* Read before the Pediatric Section, New York Academy of Medicine, December 8, 1904.

vomiting had set in after the second week, although he had been exclusively breast-fed, the condition of the bowels meanwhile varying from constipation to moderate diarrhœa, with green stools. The family history was negative; her other child was quite normal. Examination of the infant revealed nothing except extreme loss of flesh and strength; abdominal palpation was rendered almost futile by the child's continuous crying. Dietetic experiments showed that albumin-water and ordinary barley-water were regularly vomited within twenty to thirty minutes, the curdled condition of the vomitus showing approximately normal acidity of the gastric juice. According to the mother's statement, the vomiting was forcible, not merely regurgitant; that it was not complete could be demonstrated with the stomach tube, whereby several ounces of fluid could be withdrawn from the stomach. Dilute barley-water and lime-water were usually, though not always, retained. After a week of observation I was led to suspect the presence of a so-called congenital hypertrophy and stenosis of the pylorus; a positive diagnosis was, however, unattainable, for neither a tumor nor gastric peristalsis could be made out. Lavage was resorted to, with some benefit; the vomiting had been, in a great measure, abated, when the infant, rather suddenly, went into fatal collapse on October 13th. On account of the extreme marasmus, the grave diagnostic doubts, and the disputed advisability of surgical intervention in these cases, operative measures had not been counselled; it turned out that in this case no other procedure could have been of the slightest use, and that its omission was an error, as I now freely admit, although the infant most probably would have succumbed to the shock of the operation.

A partial, but sufficient, autopsy was obtained with considerable difficulty, and performed within six hours, rigor mortis being still present. On opening the abdomen, the enlargement of the moderately congested liver and spleen attracted attention; the former organ completely overlapped the pyloric region, so that the almost cartilaginous mass of the pylorus could only be palpated by working deeply underneath; it was evident that it could not possibly have been made out during life. Not counting the pyloric end, the stomach had an extreme length of 10 cm., and was distended with air and a gelatinous fluid; the walls of the organ were tense and of about normal thickness, the folds of its pale mucous membrane almost obliterated. The pylorus was 3 cm. long, of remarkably firm consistency, projecting into the duodenum like a cervix uteri; its diameter of 14 mm. was in marked contrast to the diameter of only 8 mm. of the empty, flaccid, and somewhat congested duodenum. The lumen of the pylorus barely admitted a director 2 to 3 mm. in thickness; its walls had a thickness of 6 mm., whereof the greater portion evidently represented muscle. The mucous membrane appeared perfectly normal, and was thrown into heavy longitudinal

folds which must have rendered the already narrow orifice almost wholly impervious. The intestine was normal, save for a contraction and possibly deficient development of the lower ileum. The result of the autopsy was a positive diagnosis of hypertrophy and stenosis of the pylorus; the microscopic findings I shall give later on.

The first mention in literature of this disease dates from 1788, when Beardsley[1] published a case, whose status I shall discuss presently. Beardsley's report sank into oblivion, to be resuscitated at Osler's instance a year or two ago. In 1841 and 1842 Williamson[2] and Siemon-Dawosky[3] respectively published two cases that are typical, and will always remain classical. Then follows a great gap until 1888, when Hirschsprung[6] brought the subject before the profession once more, none of the intervening reports (Landerer,[4] Maier[5]) applying to infants, but giving cases in adult subjects only. It seems strange that so characteristic a lesion and so striking a clinical picture should have escaped observation for forty-six years; at any rate, no case came to autopsy, and we shall see that no case without operative or post-mortem autopsy can be accepted without reserve.

Since Hirschsprung's communication enough has been written about this disease to fill a good sized volume; a great part of this literature is, however, uncritical, and still more purely academic; verified reports are still scanty, even with the most liberal interpretation.

Clinically and pathologically, we may divide all the reported cases of congenital hypertrophy and stenosis of the pylorus into three groups.

1. There are those that attain an age beyond early infancy, present throughout their lives more or less symptoms of obstruction, in some cases even grow to maturity, and then seek operative relief. It is evident that here the stenosis must be of a moderate degree; the pyloric lumen, as a matter of fact, measures from 5 mm. upward.

2. We have to consider those infants that present for a few weeks similar symptoms, which, however, gradually yield to appropriate diet and medication.

3. Those babies that either are promptly relieved of an almost total obturation by surgical measures, or die within a few weeks of simple inanition.

The cases coming under the first head are fairly numerous; there is Beardsley's that died at the age of five years, those of Landerer and Maier, the second one of Hirschsprung, those of Henschel,[8] more recently those of Rosenheim (Sonnenburg),[28] Hansy,[31] and so on, latterly, that of Shaw.[54] Batten's[30] case is doubtful; it improved under treatment, and died at eleven weeks of bronchopneumonia. This group need not detain us further, for, even if now and then a

case, such as the last, may not admit of classification, these patients present no urgent symptoms in infancy, and therefore afford a different picture, both clinically and prognostically, from those to follow.

The second group is also quite well represented, and is of far greater interest. I shall refer to only a few reports, namely, those of Weill and Péhu,[34] Southworth,[39] Saunders,[46] Gardner,[53] Freund,[55] Variot,[57] and Stamm,[66] but could mention many more. We can discuss the status of this series to greater advantage in connection with the third group than by attempting to dispose of it separately, and shall therefore pass on at once to the latter. It is this, the type, which chiefly interests us, and will form the body of this paper; we have here the advantage of dealing with definite and tangible data which will form a convenient starting point. I present below a tabulated synopsis of the moderate number of cases that have come to autopsy, omitting the operated cases, which are probably more suitable to the paragraph on treatment, and have not always been carefully reported. A fitting introduction to this table is Williamson's classic account, which it will be worth while to quote in full:

"The infant, a male, died at the age of five weeks. At birth it was plump, and apparently healthy, but a few days afterward vomiting came on, and the matter ejected was coagulated milk. During the last fortnight the bowels were obstinately constipated, and the child seemed to be falling off considerably in flesh, until it gradually sunk exhausted. Upon dissection, the intestines were found collapsed and empty, and all the other visceral organs perfectly healthy, with the exception of the stomach, the pyloric extremity of which felt hard and indurated, forming a remarkable contrast to the soft and yielding parietes of that viscus when emptied of its contents. Upon removing the stomach it was found that the pyloric orifice was so contracted as scarcely to admit a small silver probe, a state which might perhaps in part account for its being nearly filled with coagulated milk. Upon slitting open the pyloric orifice, it was evident that the tissues entering into the composition of the parietes of the stomach had greatly lost their normal appearance.

The mucous coat was slightly thickened, while scarcely a distinct remnant of the middle or muscular tunic was observable. On the other hand, the submucous cellular tissue was so much hypertrophied and indurated as seemingly to be the only tissue contained between the mucous and peritoneal coats."

Williamson's histological diagnosis of "scirrhus" cannot, of course, be accepted; otherwise his description is perfect. We should not even criticise the "scirrhus" too severely; many of us still speak of uterine "fibroids."

We may now pass on to our table:

No.	Author.	Sex.	Age at onset.	Age at death.	Bowels.	Tumor felt?	Dilatation		Lumen of pylorus.
							or peristalsis in vivo	p.m.	
1	Williamson,[3]	M.	3 dys.	5 wks.	Constipation.	No	No	?	2 mm.
2	Dawosky,[3]	?	4 wks.	10 wks.	"	"	"	?	3 mm.?
3	Hirschsprung,[6]	F.	10 dys.	4 wks.	"	"	"	No	2–3 mm.
4	Peden,[7]	?	3 dys.	12 wks.	"	"	"	"	5 mm.
5	Pitt,[9]	?	3 wks.	7 wks.	?	"	"	"	?
6	Finkelstein,[10]	F.	at birth	13 wks.	Constipation.	Yes	Yes	Yes	?
7	Gran,[11]	F.	2 wks.	16 wks.	Diarrhœa.	No	"	"	3 mm.
8	DeBruyn Kops,[12]	M.	at birth	7 wks.	Constipation.	"	No	"	5 mm.?
9	Thomson,[13]	M.	10 dys.	4 wks.	Moderate constipation.	"	"	"	5 mm.?
10	Thomson,	M.	4 wks.	10 wks.	Constipation.	"	"	"	1 mm.?
11	Thomson,[14]	F.	1 wk.	7 wks.	?	Yes	"	No	?
12	Schwyzer,[15]	F.	2 wks.	11 wks.	Constipation and diarrhœa.	No	"	Yes	2 mm.
13	Schwyzer,[16]	M.	?	7 wks.	Constipation.	"	"	"	2 mm.
14	Ashby,[17]	?	1 wk.	7 wks.	"	"	"	"	2–3 mm.
15	Rolleston and Hayne,[23]	M.	at birth	8 wks.	"	"	"	"	2 mm.
16	Still,[25]	M.	6 wks.	12 wks.	Diarrhœa.	Yes	"	slight	3.5 mm.
17	Still,	M.	6 wks.	14 wks.	Constipation.	"	Yes	Yes	3.5 mm.
18	Still,	M.	3 wks.	14 wks.	"	"	"	"	3.5 mm.
19	Cautley,[26]	M.	5 wks.	14 wks.	"	No	No	?	2 mm.
20	Cautley,	M.	at birth	8 wks.	?	"	"	No	closed.
21	Pritchard,[32]	M.	3 wks.	9 wks.	Constipation.	"	Yes	Yes	3 mm.
22	Cautley,[33]	F.	at birth	12 wks.	Constipation and diarrhœa.	"	No	"	2 mm.
23	Rolleston and Crofton-Atkins[35]	M.	2 wks.	8 wks.	Constipation and diarrhœa.	"	"	"	1–2 mm.
24	Blackadder,[42]	M.	2 wks.	10 wks.	Constipation.	"	"	"	?
25	Cautley,[43]	?	?	8 wks.	?	?	?	"	1–2 mm.
26	Saunders,[46]	F.	2 wks.	16 wks.	Diarrhœa.	Yes	Yes	"	2–3 mm.
27	Nordgren,[47]	F.	at birth	11 wks.	Constipation.	No	No	No	4 mm.
28	Nordgren,	M.	3 wks.	7 wks.	"	"	"	Yes	5 mm.
29	Riviere,[52]	M.	2 wks.	8 wks.	Constipation and diarrhœa.	"	"	No	?
30	Freund,[55]	M.	?	9 wks.	?	?	?	?	?
31	West,[56]	M.	3 dys.	5 wks.	Constipation and diarrhœa.	Yes	Yes	Yes	?
32	Cautley,[62]	M.	3 wks.	14 wks.	Constipation and diarrhœa.	"	"	"	1–2 mm.
33	Cleveland,[63]	M.	2 wks.	12 wks.	Constipation.	No	"	"	5 mm.
34	Dorning,[65]	M.	at birth	11 wks.	Constipation and diarrhœa.	Yes	No	slight	2 mm.
35	Wachenheim,	M.	2 wks.	7 wks.	Constipation and diarrhœa	No	"	slight	2–3 mm.

A few explanatory words may be added. The vomitus was usually acid; free HCl was found in a few cases, bile in Nos. 13 and 26. In many cases the palpated tumor was almost certainly not the pylorus, but the tensely distended fundus. The lumen is often reported as admitting "a fine probe," "a No. 6 catheter," "the blade of a pair of forceps," and so on. I have endeavored, for the sake of easy tabulation, to reduce all such statements to millimetres.

The average age at onset is two weeks; at death nine weeks and a half. In view of the remarkable uniformity of the above statistics, we may safely regard every case that survives beyond four months as outside of this clinical group. Indeed, it stands to reason that an almost or quite complete occlusion of the gastrointestinal aperture is incompatible with longer life.

Males predominate in the ratio of 2.5 to 1. Most of the infants were breast-fed; improper food played no causative part. The

intensity and frequency of vomiting were directly proportional to the quantity and consistency of the ingesta, as happened in my case. The acidity often noted in the vomited matter was probably not often due to free HCl, but in most cases this point was not determined. Biliary vomiting occurred a few times; it seems inconsistent with the anatomical conditions, and might give rise to diagnostic doubts, as in Schwyzer's second case. Uncertainty is also bound to arise when diarrhœa and green stools are present, especially during the warmer months. It requires a week or so of observation to clear away doubts in all but the typical cases, and often the diagnosis will not advance beyond probability. For my part, I would venture farther, and assert that most cases remain unrecognized and unreported, being passed by as mere gastro-intestinal catarrhs, and I am supported in this opinion by such authorities as Cautley.

The palpation of the pylorus, as suggested above, is an uncertain quantity, and is frequently rendered difficult or impossible by the almost constant crying of the infant, with resultant tension of the abdominal parietes. Opinions vary widely as to the presence or absence of pain; while the crying may in part be due to hunger, the violent contractions of the gastric muscle, as sometimes evinced by visible peristalsis, and the forcible vomiting must cause great distress. Peristalsis and dilatation were visible usually only when the stomach was well filled, and sometimes only in the terminal stages of the disease, in many cases not at all. This appears quite natural, since the recorded post-mortem measurements of the stomach rarely show more than moderate dilatation, the extreme length rarely equalling 12 and often not exceeding 9 cm., and the walls of the viscus being but very slightly changed, if we except the pyloric end.

In view of the usual off-hand designation of this affection as congenital, it is curious that most cases show no symptoms during the first week or two, sometimes even up to six weeks, the onset being in general quite abrupt. This point is most perplexing and not readily explainable; we should infer from this circumstance alone that the question of pathogenesis is difficult to answer; as a matter of fact, clinicians have been, for some time, grouped into three factions, two holding opinions diametrically opposed, while the third occupies a middle position.

Dating from Thomson's reports, many cases have been recorded as "congenital spasm of the pylorus," often such as have not come to autopsy or have apparently recovered; some, however, like those of Thomson himself, of the true Williamson type. Still,[24] whose anatomical studies are invaluable, is convinced that the chief element in this disease is spasm, that the hypertrophy is secondary and not of a high degree, the relatively short duration of the affection admitting of only moderate muscular overgrowth, that serious and

permanent changes are inconsistent with the reported cures, and that the narrowing of the pyloric lumen, as found post-mortem, is often very moderate. Still's own cases, given in the above list, are, unfortunately, not among the most typical; the lumen in these cases was indeed "3.5 mm. or more." The onset was unusually late, and the duration of life beyond the average; in fact, they form the transition, if one exists, to cases such as Batten's, which we have been compelled to exclude from consideration here. Pfaundler,[19] writing shortly before Still, goes farther; he claims that the pylorus of an entirely normal stomach may remain contracted post-mortem, thus assuming the consistency of an annular tumor with apparently hyperplastic walls, narrowing the lumen almost to obliteration; secondly, he insists that the anatomical and histological pictures, presented by Hirschsprung and others as pyloric stenosis, agree perfectly with the aforesaid systolic stomach; thirdly, he instances the reported recoveries from similar symptoms, refers to our ignorance of any anatomical basis for this affection, and concludes that we are dealing with a functional spasm alone. Schmidt,[44] in connection with his operated case, to be noted farther on, agrees entirely with Pfaundler; his case is, however, a more than doubtful one, the lumen of the pylorus measuring over 5 mm. Freund[55] goes a step farther; asserting that the spasm is probably neuropathic, and associated with or even caused by hyperchlorhydria. Hyperchlorhydria in infants had recently been discovered by Knöpfelmacher,[45] and from our knowledge of certain spasms in the adult Freund's view might appear plausible, but for the fact that free HCl was not always present in the vomited matter; even so, free HCl without quantitative data does not imply hyperchlorhydria by any means.

I have gone into the spastogenic theory somewhat deeply, because it does not represent the prevailing opinion. Stern,[20] the first to operate for this condition, considers it almost wholly an anatomical lesion; his article being the occasion for the just quoted statement of Pfaundler. Meltzer,[21] whose case was the second to be operated upon, makes a strong plea, from the standpoint of the pathological anatomist, for the hypertrophic (hyperplastic) theory; he insists that a spasm, lasting a few weeks only, cannot cause the excessive muscular development found in these cases, and that the concomitant increase of the submucous connective tissue shows hyperplasia and not merely conpensatory hypertrophy; in the severe cases, that will hardly admit of the passage of a probe, pyloric spasm can contribute but little to a condition already intolerable. Cautley and Dent,[50] the former of whom has seen one-eighth and reported one-fourth of all recorded cases, take the same position; the remarkable uniformity of Cautley's unique experience must be allowed to have especial weight. A strong point in favor of the observers of the second group is, that their stand is not incompatible with a compromise in the direction of the following.

We thus pass on, quite naturally, to the third group, who are prepared to accept both propositions. They believe, and I feel compelled to agree with them, that there is one condition that consists in an overgrowth of the muscular and submucous coats of the pylorus, narrowing the orifice of that viscus, and aggravated by a tonic contraction which is in all probability due to the abnormal innervation that usually accompanies muscular hypertrophy. They also admit a second condition, a pyloric spasm pure and simple, resting possibly on the hypothesis of Freund, and associated with some slight secondary muscular hyperplasia. It is evident that the prognosis of the former lesion is bad, and of the latter fairly good. Finally, we must reckon with the possible occurrence of intermediate or transitional states. The evidence on this point is incomplete, but it certainly is curious that cases like Still's[25] and Nordgren's,[47] with moderate stenosis, have a pyloric wall only about 4 mm. in diameter; while severe cases, such as Schwyzer's,[16] Cautley's,[51] and mine, have a pyloric wall of far greater thickness. While, from the clinical aspect, this matter must remain, for the time being, academic, it may, eventually, have important and interesting pathological bearings. Clinically, we can only consider two distinct affections, whose symptoms are almost precisely alike, but whose anatomy, course, and prognosis differ very widely.

Schotten,[61] in his recent excellent monograph, plainly accepts this view. When he says that the prognosis of hypertrophic stenosis is hopeless, he clearly considers all reported cures to apply to cases of simple spasm; in other words, he assumes the existence of two distinct diseases. This does not, of course, imply that all cases of pyloric spasm recover, but from the scarcity of autopsies, at most a small minority of those in our table, a fatal result must be unusual.

Whether the pyloric abnormality is a deformity or a hyperplasia in the sense of a new-growth cannot be decided. There is some evidence in the former direction. Ashby's second case, excluded from my table, was an infant a few days old, that died after an operation for atresia of the anus; the pyloric orifice was found to be quite impervious, the organ itself much thickened. Two similar cases are reported by Hammer;[27] related to these are several records of congenital occlusion of the duodenum. I cannot determine whether or not these facts apply here; the question seems to me an open one. As to the theory of a new-growth, it has so far nothing to sustain it, yet need not be absolutely rejected on that account, since the whole subject of benign and other new-growths is still shrouded in mystery. We know of no strictly analogous lesion in any other organ.

Let us pass on to anatomical details, prefacing them with a few remarks concerning the not generally known normal conditions. Still[25] and Pfaundler[18] have made valuable contributions to our knowledge of the dimensions of the normal infantile pylorus, which

it will repay to give here in condensed form. Still's results are not very precise, though very probably quite correct; he states the average total thickness of the pyloric wall at 2.5 mm. in infants of about six months, with an extreme range of about a third more or less; the minimum for the normal lumen he sets at 4 mm., a rather low and conservative estimate. Pfaundler gives us detailed measurements of the pyloric orifice, 6.3 mm. at birth, 7.3 mm. at two months, 8.5 mm. at six months, and 10.5 mm. at twelve months, all averages, of course. According to both authorities, a lumen of over 5 mm. at two or three months, as frequently recorded in our tables, can hardly amount to a serious anatomical lesion, as should, indeed, be obvious without much investigation; the importance of this point will be emphasized in discussing treatment. Still's measurements seem to settle conclusively that a pyloric wall over 4 mm. thick is pathological. So far, all is clear.

When the orifice of the organ measures less than 3 mm., the attendant spasm of the hypertrophied muscle seems sufficient to cause almost complete and permanent closure; that this is, however, not quite total, is shown by the passage of thin fluids, occasional biliary vomiting, and by the almost invariable presence of fecal matter in the bowel, sometimes even a diarrhœal condition. Freund made some interesting pressure experiments, showing the resistance of the pyloric sphincter to abnormally high hydrostatic pressure, not a drop passing through; this circumstance had been somewhat cursorily noted by a number of previous observers. It seems that these results depend in part on post-mortem rigidity, in part on valve action of the marked folds of the mucous membrane; the valve action would, naturally, increase with abnormal hydrostatic pressure, yet might not exist to the same degree during life. That this aspect of the case has some merit is shown by the clinical fact that vomiting is far more apt to follow a full meal than teaspoonful doses of thin fluids, also by the undoubted temporary relief afforded by the stomach tube.

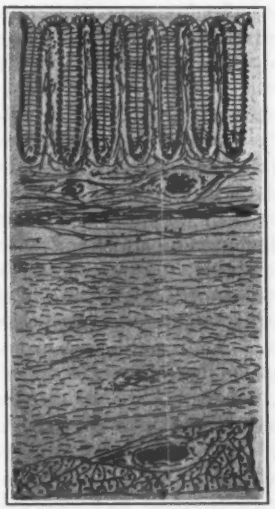
I proceed to the histological findings in this disease, beginning with a study of my own case, and comparing this with the, save for a gastritis, normal stomach of an infant of the same age. For the latter specimen I am indebted to the kindness of Dr. S. V. Haas.

In the mucosa and its glands there is nothing abnormal. The submucosa is of about twice the normal thickness, with considerable round-cell infiltration. The muscular coats are enormously hypertrophied; the circular coat has four to five times the normal thickness, and the overgrowth of the longitudinal muscle is proportionately nearly as great. In the muscular coats (chiefly in the circular) there is also a moderate amount of inflammatory reaction, but, perhaps, not more than one would expect with so enormous a hypertrophy in infantile tissue. There is no change in the bloodvessels out of proportion to the excessive development of the other tissues.

The accompanying illustrations will fully illustrate the disturbed relations and measurements of the various parts in my case. Both sections are taken from the thickest portion of the viscus.

It is interesting to compare the findings of Finkelstein,[10] who claims that the hypertrophy is chiefly in the longitudinal coat. Most other observers have laid stress on the increase of the circular coat; this seems to be a smaller error in the opposite direction. The hypertrophy of both layers in my case is so evident that it must be considered the usual picture. Upon reflection, we readily see that it could not be otherwise; to overcome the abnormally firm closure of the pylorus by the circular layer, there must be a compensatory hypertrophy of the longitudinal coat, whose function is

FIG. 1.



Normal pylorus at seven weeks (cross-section). × 25.

FIG. 2.

Case of Thomas M. Pylorus (cross-section). × 12½.

that of dilatation and propulsion. The hyperplasia will naturally be somewhat greater in the circular layer, where it is primary. In accordance with this is the more abundant evidence of interstitial inflammation in the circular layer, where we are dealing with the primary hyperplasia; in the at least partly compensatory overgrowth of the outer coat, round-cell infiltration is not so marked.

The theory of spasm cannot, of course, apply to such a case as this at all. Aside from the very plainly visible new-growth of tissue, the thickening of the longitudinal coat does not fit into such an hypothesis. It hardly seems worth while to go into this matter

deeply, for the bulk of the pylorus in my case is at least three times as great as normal, and the defender of the spastic theory can be at once confronted with the demand that he account for the presence of the redundant two-thirds of tissue. If the thickening of the pylorus were due to spasm the organ would be shortened, its total volume remaining unchanged; in my case the length as well as the diameter of the pylorus is increased, as can be seen by its cervix-like projection into the duodenum.

The rarity of the disease has undoubtedly been exaggerated. When a single English observer can furnish a long series of cases, the failure of French authors to produce a single one is explainable in only one way. The very peculiar distribution of the available records convinces me that hundreds of cases have been overlooked within the last ten years. With an obscure disease this would not cause astonishment; in view of the exceedingly simple anatomical picture here presented it is most remarkable. We have every reason to expect an enormous increase of material within a brief period.

The diagnosis, in many cases, is not easy; in summer the possible combination of pyloric stricture with a digestive disturbance will almost always prove puzzling, especially as, among the poorer classes, the histories given by the mothers are unreliable. I have only to mention the fact that in my service the anamnesis of diarrhœa is rarely volunteered, but has to be elicited by leading questions; we all recognize the overpowering tendency to answer leading questions in the affirmative. The key-note in this affection is the combination of obstinate vomiting, equally obstinate constipation, and gradual loss of weight; an important point in the vomiting is its violence and close dependence on the ingestion of food, the quality of which is almost immaterial; meanwhile the appetite, as might be expected, is ravenous; this complex of symptoms is highly characteristic. I have referred sufficiently to such matters as visible dilatation and peristalsis, the doubtful palpability of the pyloric tumor, the normal condition of the peptic function as revealed by the stomach tube, and need not weary you with a repetition of these data.

Diagnosis is but the preliminary step to prognosis; while the academic discussion of the latter is easy, the problem in the individual case becomes almost unsolvable. If Pfaundler's opinion is correct, most cases should recover. I have sufficiently stated my reasons for excluding all cases of mere pyloric spasm from this discussion; in the cases of true stenosis, the outlook is for a rapidly fatal issue. The only demand on the diagnostician, therefore, is to establish an anatomical obstruction; this has hitherto been feasible only by prolonged observation and *ex juvantibus*, whereby the opportunity for relieving the patient is apt to be lost by undue delay. Thus, the clinician's task is trying and embarrassing; time

is required for study, yet a true stenosis of the pylorus in a young infant does not permit much temporizing.

As it is impossible, in a particular case, to determine at once whether mere spasm or organic obstruction is playing the chief role; and since operative intervention, as we shall see, entails a high proportionate mortality, it would appear in order to begin by assuming the presence of the milder condition, from which recovery is the rule, and treat the patient accordingly. In this connection Freund's method is of great interest, as it resulted in a permanent cure of one-third of his cases, while another third died only in consequence of intercurrent affections after apparent cure. His treatment consists in giving Carlsbad (Mühlbrunnen) water in addition to the usual diet—*i. e.*, breast-milk, later on whole milk, resorting to surgery only when this treatment fails. Although his procedure rests on the above-mentioned theory of spasm from hyperchlorhydria, which he acknowledges to be often absent, yet his results warrant further trial. Some benefit also seems to be derived from lavage; at least, it diminishes the strain on the infant organism, caused by frequent and violent spells of vomiting. I would also refer to the advantage of giving small and frequent meals of highly diluted albumin-water and barley-water; this plan naturally failed in my case of extreme stricture; in spastic cases it should prove vastly beneficial, if resorted to before grave exhaustion has supervened.

The procedures mentioned embrace about all that can be recommended in the way of medical measures, and I consider it an error to continue them beyond two weeks, unless the infant begins to gain in strength and especially in weight, which latter should be carefully controlled. Further than this, all treatment must be strictly surgical, as affording the only chance, though not a very brilliant one.

I shall begin the consideration of the surgery of this disease by once more giving all available reports in tabular form:

No.	Author.	Age.	Operation.	Result.	Lumen of pylorus.
1	Stern,[20]	6 weeks.	Gastroenterostomy.	Fatal in 12 hrs.	2 mm.
2	Meltzer (Meyer),[21]	6 "	Gastroenterostomy (Murphy button).	" 30 "	1–2 mm.
3	Meyer (Adler),[22]	11 "	Gastroenterostomy.	" 16 "	2–3 mm.
4	Abel (Kehr),[29]	8 "	"	Cured.	2–3 mm.
5	Löbker,[27]	11 "	"	"	?
6	"	7 "	"	Fatal.	?
7	Kehr,[26]	8 "	"	Cured.	?
8	Fritsche,[28]	?	"	"	?
9	Lange (Braun),[60]	10 weeks.	Pyloroplasty.	Fatal.	1–2 mm.
10	Nicoll,[41]	6 "	Divulsion.	Cured.	?
11	Schmidt,[44]	8 "	"	"	5–6 mm.
12	Cautley and Dent,[50]	8 "	Pyloroplasty.	Cured; died 13 weeks after of intestinal disorder.	?
13	" " "	6 "	"	Cured.	?
14	Burghard,[51]	8 "	Divulsion.	"	?
15	Murray,[51]	18 "	Pyloroplasty.	"	?
16	Stiles,[51]	9 "	Pylorectomy.	Fatal.	?
17	"	5 "	Gastroenterostomy.	"	?
18	"	7 "	Divulsion.	Cured.	?
19	"	4 "	"	Fatal.	?
20	"	11 "	"	Cured.	?
21	Graanboom,[48]	3 "	Pyloroplasty.	Died 6 wks. later from inanition.	2 mm.
22	Trantenroth,[49]	7 "	Gastroenterostomy.	Cured.	2–3 mm.
23	Freund,[55]	10 "	"	Died 19 weeks later from intestinal hemorrhage.	?
24	Greeff,[58]	?	"	Fatal.	?
25	Gallant,[59]	5 weeks.	"	Fatal in 6 hrs.	2 mm.
26	Mackay,[60]	20 "	"	" 36 "	3 mm.
27	"	6 "	Divulsion.	" 22 "	?
28	Schotten,[61]	5 "	Gastroenterostomy.	Cured.	?
29	"	6 "	"	Fatal in 20 hrs.	?
30	"	20 "	Gastroenterostomy (uncompleted).	Fatal.	3 mm.
31	McCaw and Campbell,[64]	?	Pyloroplasty.	Fatal in 6 days.	3 mm.!
32	Grisson,[67]	9 weeks.	Divulsion.	Cured.	2 mm.

(Cases 14 to 20 reported very briefly by Cautley and Dent.)

SUMMARY.

Operation.	Cured.	Fatal.	Total.	Mortality.
Gastroenterostomy (suture)	6	10	16	62 per cent.
" (Murphy)	0	1	1	100 "
Pylorectomy	0	1	1	100 "
Pyloroplasty	3	3	6	50 "
Divulsion	6	2	8	25 "
Total	15	17	32	53 "

This summary, which gives a highly favorable result for divulsion, is in great need of correction for inaccuracy. Thus Schmidt's case is probably not one of true stenosis, and might have recovered without operation; in Case No. 31 the operation seems to have been positively harmful, but in view of the before-stated diagnostic difficulties, the surgeon is in every way excusable; cases 14 to 20, mere private communications to Cautley, and published without comment, are too briefly reported to be worthy of inclusion. Omitting these 9 cases, our summary is as follows:

Operation.	Cured.	Fatal.	Total.	Mortality.
Gastroenterostomy (suture) .	6	9	15	60 per cent.
" (Murphy)	0	1	1	100 "
Pyloroplasty	2	2	4	50 "
Divulsion	2	1	3	33 "
Total . . .	10	13	23	57 "

Even these figures are probably too favorable; in many more cases, as shown in the table, the pyloric lumen is not reported; some of these may be merely cases of spasm. Of the 11 positive cases, 8 died, 73 per cent.; this is probably near the true proportion, for in this series, as in all surgical ones, many unfortunate cases never appear in print, while successful ones rarely escape notice. I may mention that Freund's case seems to have died from the tearing out of a suture; the result is therefore reckoned among the failures.

The summary, given above, seems to show about equally good results for all procedures, except the Murphy button method, which has not had a fair trial. Dr. Willy Meyer has kindly favored me with a communication concerning his case, in which the Murphy button was used; the button employed was far too large, and he has had no opportunity to employ the small ones he has ordered for a possible future occasion; he is not, however, inclined to regard that method very favorably. Not being a surgeon, I forbear descanting on the relative merits of the other methods; moreover, the question is still open, and can be settled only by further trials. It will be noted that German surgeons favor gastroenterostomy, the other methods being preferred in England. As to divulsion, the fatal cases seem on autopsy to show a rapid recurrence of the stenosis, leading one to suspect that the cures embraced mostly spastic cases without serious organic obstruction.

I feel that, in spite of every effort to give a painstaking and exhaustive analysis of the subject of hypertrophy and stenosis of the pylorus in infants, the general impression is one of uncertainty, and in many points quite confused. I also feel, much to my regret, that this impression is only too fully warranted by even a careful summary and intelligent discussion of the facts in our possession.

ADDENDUM. Shortly after the above was written there appeared a monograph by Ibrahim (*Die angeborene Pylorusstenose im Säuglingsalter*, Berlin, 1905), in which he reports 7 cases, 4 of which recovered, 1 died at eleven months, and 2 were unsuccessfully operated upon (gastroenterostomy) at eight and ten weeks respectively. Ibrahim obtained a complete autopsy of one of these; the lumen of the pylorus was 4 mm., and the hypertrophy affected almost wholly the circular layer, which had 2.3 times the normal thickness; the case thus resembled those of Still. Ibrahim proposes a new theory, namely, that there is an anatomical stenosis in early infancy, which undergoes spontaneous cure by compensatory hyper-

trophy in most cases; these cured cases cannot therefore be referred to simple spasm. Observe, however, the differences from such cases as those of Cautley and myself: first, the much wider, in fact, almost adequate, lumen; second, the almost total absence of hypertrophy in the longitudinal muscularis. Ibrahim's case is assuredly not typical. His theory is, however, worth mentioning, even if it rests, so far as our knowledge goes, on anatomical premises which have not been demonstrated and may be without foundation. It seems far-fetched to treat of this affection as a quasi-normal process; on the other hand, the possibility of a spontaneous cure of very moderate degrees of muscular hypertrophy cannot be rejected finally, as matters now stand.

References.

1. Beardsley. Archives of Pediatrics (reprinted), May, 1903.
2. Williamson. London and Edinburgh Monthly Journal, 1841.
3. Siemon-Dawosky. Casper's Wochenschrift, 1842, No. 7.
4. Landerer. Inaugural Dissertation, Tübingen, 1879.
5. Maier. Virchow's Archiv, 1885, Bd. cii.
6. Hirschsprung. Jahrb. f. Kinderheilk., 1888, Bd. xiii.
7. Peden. Glasgow Medical Journal, 1889.
8. Henschel. Arch. f. Kinderheilk, 1891, vol. xiii.
9. Pitt. Lancet, October 24, 1891.
10. Finkelstein. Jahrb. f. Kinderheilk., 1896, vol. xliii.
11. Gran. Ibid.
12. De Bruyn Kops. Nederl. Tijdschr. voor Geneesk., 1896, vol. ii.
13. Thomson. Edinburgh Hospital Reports, 1896, vol. iv.
14. Thomson. Scottish Medical and Surgical Journal, June, 1897.
15. Schwyzer. New York Medical Journal, November 21, 1896.
16. Schwyzer. Ibid., November 27, 1897.
17. Ashby. Arch. of Pediatrics, July, 1897.
18. Pfaundler. Wiener klin. Wochenschr., 1897, No. 44.
19. Pfaundler. Ibid., 1898, No. 45.
20. Stern. Deut. med. Wochenschr., 1898, No. 38.
21. Meltzer. Medical Record, August 20, 1898.
22. W. Meyer. Annals of Surgery, 1898, vol. xxviii. p. 247.
23. Rolleston and Hayne. British Medical Journal, April 23, 1898.
24. Still. Ibid., February 11, 1899.
25. Still. Transactions of the Pathological Society, London, 1899.
26. Cautley. Medico-Chirurgical Transactions, 1899.
27. Hammer. Prag. med. Wochenschr., 1895, No. 34; and 1899, No. 3.
28. Rosenheim. Berl. klin. Wochenschr., August 7, 1899.
29. Abel. Münch. med. Wochenschr, 1899, No. 48.
30. Batten. Lancet, December 2, 1899.
31. Hansy. Wiener med. Wochenschr., 1900, No. 11.
32. Pritchard. Arch. of Pediatrics, April, 1900.
33. Cautley. Lancet, July 28, 1900.
34. Weill and Péhu. Lyon médical, December 9, 1900.
35. Rolleston and Crofton-Atkins. British Medical Journal, December 22, 1900.
36. Kehr. Verh. d. deut. Ges. f. Chirurgie, 1900.
37. Löbker. Ibid., 1900 and 1901.
38. Fritsche. Monnier's Inaugural Dissertation, Zurich.
39. Southworth. Arch. of Pediatrics, January, 1901.
40. Lange. Münch. med. Wochenschr., 1901, No. 7.
41. Nicoll. Pediatrics, February, 1901
42. Blackadder. British Medical Journal, March 20, 1901.
43. Cautley. Ibid., 1901.
44. Schmidt. Verh. d. deut. Ges. f. Chirurgie, 1901.
45. Knöpfelmacher. Wiener klin. Wochenschr., 1901, No. 51.

46. Saunders. Arch. of Pediatrics, April, 1902.
47. Nordgren. Nordiskt medicinskt Arkiv, 1902.
48. Graanboom. Herinneringsbundel, Rosenstein, 1902.
49. Trantenroth. Mitt. aus d. Grenzgeb. d. Med. d. Chir., 1902, vol. ix.
50. Cautley and Dent. Lancet, December 20, 1902.
51. Cautley. Medico-Chirurgical Transactions, 1903.
52. Riviere. Lancet, December 20, 1902.
53. Gardner. Ibid., January 10, 1903.
54. Shaw. Brooklyn Medical Journal, May, 1903.
55. Freund. Mitt. aus d. Grenzgeb. d. Med. u. Chir., 1903, vol. xi.
56. West. Arch. of Pediatrics, October, 1903.
57. Variot. Gazette des hôpitaux, June 16, 1903.
58. Greeff. Medical Record, August 8, 1903.
59. Gallant. Ibid.
60. Mackay. Intercollegiate Medical Journal of Australasia, November 20, 1903.
61. Schotten. Samml. klin. Vorträge, 1904, No. 368.
62. Cautley. Lancet, March 5, 1904.
63. Cleveland. British Medical Journal, May 7, 1904.
64. McCaw and Campbell. Ibid., June 25, 1904.
65. Dorning. Arch. of Pediatrics, September, 1904.
66. Stamm. Arch. f. Kinderheilk., 1904, Bd. xxxviii.
67. Grisson. Deut. Zeitschr. f. Chirurgie, October, 1904.

A CASE OF DERMOID OF THE FEMALE URINARY BLADDER.

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Miss H., Kansas, aged eighteen years, first consulted me in September, 1904. A brunette, rather slender and somewhat frail in appearance. Very nervous and emotional, indicating previous suffering. Without any history of definite antecedent ailments, though claiming to have always been delicate. Nothing noteworthy in the family history bearing on her present condition. Some five years since she began to observe a painless urinary frequency. It was both diurnal and nocturnal and continued, varying in intensity, until one year ago, when dysuria became a prominent symptom. From that time until she came under observation periods of intense suffering, irregular as to time and duration, were noted. After a paroxysm of cystospasm and tenesmus, relief followed the expulsion of a small calculus. Neither at these periods, nor during the intervals, was there any hæmaturia. As near as could be learned, these extrusions averaged a semi-monthly repetition. The calculi were not voided into the vessel with the urinary stream, but it was alleged that they were always removed from the vulva by the patient.

A considerable number of these concretions were preserved by the patient and presented for identification at the first consultation. The smallest weighed 5 cgm.; the largest 5 dcgm. Their basic shape was pyriform, at times resembling a club; quite a number of them preserving this basic form were curved, approaching a comma, and at times an interrogation point in contour. Occasionally they described a sigmoid.

From the apex of most, if not all, there protruded a hair with a terminal investing root sheath. The color of the hirsute nucleus was that of those upon the bearer's head and mons.

The urine was large in quantity and low in gravity, with an abundance of pus cells in the sediment. The latter were the only adventitious elements worthy of remark.

The patient was very sensitive and hyperæsthetic, yet under local anæsthesia a cystoscopy became possible. In passing, it may be observed that the provisional diagnosis of dermoid was suggested. A most tenable objection to this view was that of hysterical introduction. This was entertained, but rejected. Though recognizing the urinary bladder as a favored receptacle among these neurotics, in this instance, at least, we were able to dismiss this from consideration, for reasons to appear later on.

The cystoscope revealed a congested and chronically inflamed mucosa, and lying in the trigone was a good-sized stone, weighing 19 gm. It was irregularly spherical; beyond and above this three smaller calculi, approximating the forms already described, and each containing a hair, about which clustered the slender concretions, were readily discernible. The calculi were phosphatic in composition. Since the largest was not subjected to cleavage, the character of its nucleus remains uncertain.

Beyond and to the right of the right posterior angle of the trigone, seemingly some distance from the right ureteral ostium, a projection from the mucosa indicated the presence of a tumor. Its exact size, color, form, and relations could not be noted at the time, owing to a vesical intolerance spastically crowding the instrument.

The patient being prepared in the usual manner, a general anæsthesia was administered. Thoroughly dilating the urethra, an effort was made to drag the stone through the expanded canal. This proving practically impossible without running the risk of a subsequent incontinence, and not wishing to crush the specimen, the suprapubic route was substituted.

Removing the calculi, the dermoid was located and with some difficulty seized, brought into view, and excised. It proved to be no larger than the half of a very small raspberry. Its mucous aspect resembled the former, both to the touch and in appearance. Under the loop some seven or eight protruding hairs were visible; indeed, if viewed in a proper light, they could be readily seen by the unaided eye.

The patient made an uneventful recovery. Cystoscopy, done at leisure and without local anæsthesia prior to her dismissal, disclosed the bladder with a minimum of hyperæmia. The former seat of the fetal inclusion was plainly visible; a very small, stumpy, circular disk marked its former site.

Sections of the "growth" were made by Prof. Frank J. Hall, pathologist to the Kansas City Medical College, a description of which is herewith appended.

Judging from the scant literature on the subject of dermoids in this locality, they may be regarded as among the surgical curiosities, and yet, in view of all the available data in this case, diagnosis was easy, even without the aid of instruments of precision.

It is evident that until adolescence the presence of an ectodermic alien might remain unresented. As development proceeds, however, the growth and shedding of the hairs would soon usher in a disturbance. The history of our case proves this assumption corroborative.

As the hairs grew and projected into the urinary reservoir, calcareous deposition from its excrementitious contents must have been greatest upon their distal ends. The latter being exposed longest, one can readily apprehend the tendency to club forms. For the same reason, owing to increasing and increased weight, the peculiar curvings described were quite natural. Again, the root sheath always presenting at the apex and thinnest portion of the calculus, plainly indicated a short exposure to the decomposing urine. The stone evidently fell and escaped as soon as it reached maturity.

The apical root sheath negatived the always plausible theory of hysterical perversion. In the latter event, more than likely, the deposition would have proceeded in a regular manner, forming ovals or spheres, and, if retained for some time, facets, the result of attrition, would have been present.

However frequent these fetal inclusions or rests may be in other parts of the body, they are certainly very rare in the urinary bladder.

Quite a number of recorded instances occurring in the neighborhood of the ureteral opening contained, among other elements, muscle and cartilage cells, as well as sarcoma cells. These rather suggested the character of a neoplasm, and from their nature and location probably originated in the Wolffian body.

Since hæmaturia at some time in the history of a vesical neoplasm is among the most characteristic, if not most prominent symptoms, its absence in our case was exceedingly noteworthy. It was doubly so after the cystoscope revealed the presence of a tumor. In the face of both the positive and negative testimony, it would have been impossible to escape the conclusion that this must be a dermoid.

Suppurating dermoidal evacuations by the vesical route are not uncommon. This is an entirely different matter and cannot be classed with the subject under discussion.

We are all aware that a small percentage of vesical calculi is subject to relapse after removal. The usual causes are well understood. Is it not possible that this may be an occasional factor, some element in the ectodermic tissue forming a nucleus for a renewal of the concretion?

Technically, no doubt, the entire operative procedure was feasible *via* the urethra. But whether the valuable specimen forming the basis of this contribution could have been secured intact is not so certain.

DR. HALL'S REPORT.

The following description of the bladder "tumor" presented September 15, 1904, for microscopic examination is respectfully submitted:

Gross Description. The tumor is shaped like a sphere vertically depressed. In horizontal direction the tumor measures 8 mm.; in vertical, 5 mm. It presents at the point of attachment to the bladder a small, fibrous pedicle about 1 mm. in diameter.

The color of the tumor is light pink, and in consistence elastic and soft. The surface is marked by many small dimples and rugosities. Under low magnification, or even naked-eye inspection, one makes out, emerging from the dimpled spots on the surface, rather fine hairs. One of these had a considerable deposit of urinary salts about its free end.

Microscopic Description. Sections were cut vertical to the great diameter of the tumor. Under low power the growth presents a covering of squamous epithelium resting upon typical dermal structures.

The covering epidermis shows a well-developed stratum granulosum and lucidum. The interpapillary plugs of epithelium are rather irregular in both size, shape, and distribution, but otherwise normal.

Connecting with the surface are several deep and irregular depressions lined with epidermis, and receiving at their bottoms the orifices of sebaceous glands and hair follicles. The sebaceous glands are large and typically formed. Here and there in the section are presented variously cut hair follicles and sebaceous structures.

Deeply situated in the section are several fairly large aggregations of cells which present, on close examination, the structure of glandular epithelium. The basal row of cells in these masses are tall, columnar, narrow at the free ends, and somewhat ragged. The nuclei are round and vesicular and placed near the bottom or base of cell. The cell cytoplasm stains very lightly with eosin and seems to be filled with clear, spherical spaces, which I believe to be the cavities left from absorption of fat by ether in the preparation of the sections. In other sections, typical areas of tubular glands present in variously cut sections. Here the epithelium is tall, columnar, and

presents the features distinctive of the axillary sweat glands. In the close neighborhood of these glands are many irregular spaces lined with double layers of pavement epithelium. The lumina of these spaces contain an unstained granular matter, evidently coagulated secretion. At a few points these irregular channels can be traced to the masses of gland tubules, and are evidently very tortuous and dilated ducts.

The connective tissue throughout the growth is disposed similar to that of the normal corium and constructed of the same elements. The growth is well supplied with veins and arteries whose walls are apparently somewhat thicker than vessels of corresponding size in the normal corium. In the papillary portion of this corium are areas of round cells. Here is noted frequent nuclear degeneration; the nuclei being drawn out into various bizarre threads and clumps of intensely staining chromatin.

Withal the growth is a typical dermoid.

TWO CASES OF ULCERATIVE PERFORATION OF THE RECTUM ORIGINATING FROM A GONORRHŒAL SALPINGITIS.

By Josef Otradovec, M.D.,
of Chicago.

(From Prof. Chiari's Pathological-Anatomical Institute, Prag.)

The ordinary conception of the pathological anatomy of gonorrhœa is that it is a superficial process confined mainly to mucous membranes. But the gonococcus is sometimes capable of penetrating tissues and there producing abscesses and even ulceration. Of especial note is the research of Wertheim,[1] who showed the gonococcus to be present not only in the pus, but also in the tissues of a pyosalpinx. Wertheim found, by using the staining method of Kühne (carbol-methylene blue), or even by staining with a simple methylene-blue solution, absolutely characteristic gonococci, not only in the epithelium, but also localized in the uppermost layers of the connective tissue, far more often lying in the cells than in the intercellular substance.

Thus, also, Frisch[2] found, in examination of microscopic sections of a rectal mucosa, pieces of which were extirpated both during life and after death, that in places the cylinder epithelium and partly the rectal mucosa itself, with the superficial portion of the crypts of Lieberkühn, were wanting, so that even macroscopically distinctly visible ulcers were seen. In the crypts, as well as in the connective-tissue spaces between the crypts and in the partly infiltrated mucosal connective tissue up to but not into the muscular coat, there were

gonococci massively found, which were in great numbers, both in the pus cells and lying free.

This pathological-anatomical finding is of double interest. Firstly, it shows the intercellular and intracellular wandering of the gonococci in the tissues, and, secondly, that in this case the gonococci had led to an ulcerative process.

Kraus[3] examined eight uterine pus tubes obtained operatively. In one of these gonococci were found in all the layers of the tube wall. The pus from the tube showed, both microscopically and culturally, a pure gonococcic infection. This finding substantiates Wertheim's statement that the gonococcus alone can be held responsible for inflammatory processes in the tubes and the peritoneum.

Wertheim,[4] again, in examination of tissue from a urinary bladder infected by gonorrhœa, found the gonococcus in capillaries and precapillary veins, which he thought they entered by penetrating their walls.

This finding is important, in that it explains the method of general infection by the gonococcus, and thus the gonococcus may be contemplated as only in a degree different from the ordinary pus microbes (Kiefer).[5]

The gonococcus cannot only penetrate tissues, but may lead to abscess formation in them. Menge[6] had found a pure culture of gonococci in an abscess in the wall of the uterus.

But the gonococcus can lead to purely ulcerative processes, as was mentioned in the Frisch case. Jullien[7] calls attention to the finding that, as in the rectum, thus also upon other delicate mucous membranes, the superficial tissues may be exfoliated, owing to the gonorrhœal process, leading even to necrotic ulceration.

The cases of purely ulcerative process called forth by the gonococcus are rare, and, moreover, are exceedingly difficult to demonstrate conclusively. It is not alone sufficient to find a purely gonorrhœal vaginitis, nor even a salpingitis, but the gonococcus should be demonstrated in the tissues themselves. This is difficult, owing to two reasons.

The first is that the tissues must be fresh (Kiefer[5]), owing to the fact that the gonococcus is very short-lived and in a short time disappears. In a few days the gonocoecus is to be found only in involution forms, which are very difficult to demonstrate, or are not at all demonstrable in the tissues, as involution forms scarcely stain. For this reason the finding of the gonococcus in tissues does not depend upon the method of staining, but upon the condition of the tissues to be examined. If the gonococci are young and therefore easily stained, their demonstration is affected easily and by all staining methods, even with the ordinary watery solution of methylene blue; if they are old and in involution, they cannot be safely demonstrated by any method.

Again the difficulty arises, that after ulceration mixed infection

easily occurs, and, therefore, other bacteria besides the gonococcus are seen in the tissues, thus apparently vitiating the results of the examination.

The published cases of ulceration due to gonococci are few, and even some of these, for above reasons, are doubtful. Cohn[9] published a case of rupture of a gonorrhœal salpingitis into the intestine. But in this case a purely gonorrhœal process could not be assumed. The following case, however, is positive. Kiefer[10] mentions a case operated upon by A. Martin, similar to the above, which convinced him that an ulcerative process due to gonococci alone is possible. In this case there was a bilateral gonorrhœal pyosalpinx. Both from the cervix and after operation from the pus in the tubes, microscopically and culturally, gonococci alone were demonstrated in the pus in pure culture. At the operation the tubes were so adherent to the rectum that it was impossible to separate them without doing damage to the rectal wall. A small piece, the size of a large coin, about one and one-half the thickness of the normal intestinal wall, adhered to the removed tube. On microscopic section through this musculature there was found, along the blood and lymph vessels, massive infiltration, consisting mostly of polynuclear leukocytes, in which many gonococci were found.

Kiefer thought that this finding makes it highly probable that without the operation in this case also, and in no distant time, a perforation into the rectum would have occurred.

Two cases came to autopsy at Prof. Chiari's institute, which point very probably to the conclusion that an ulcerative process due merely to gonococci was present.

The first case had the following clinical history: O. A., aged eighteen years, a servant, was admitted to the clinic of Prof. von Franquè at the Allgemeinen Krankenhaus of Prag on July 4, 1904, complaining of swelling of the abdomen and obstipation.

At the gynecological examination a discharge from the vagina was noted, pain and infiltration of the parametrium, and a diagnosis of parametritis was made.

On the next day, July 5th, she was transferred to the surgical clinic of Prof. Wölfler. There a diagnosis was made of acute intestinal obstruction, and an immediate operation undertaken.

Status Præsens. Of medium height, strong bone structure, well-developed musculature, and fair panniculus adiposus. Mammæ well developed. Percussion of the chest showed no abnormalities. Pulse rapid, small, and of increased tension. Temperature of skin normal.

Abdomen swollen, cone-shaped, mostly at the region of the navel. On pressure great pain was elicited. Percussion tone was everywhere tympanitic. The area of liver dulness somewhat decreased. Pain distinctly greater in the lower regions of the abdomen.

Operation, July 5th. Incision in the median line 10 cm. long. After opening the peritoneal cavity the small intestines, which were

dilated to the size of a man's fist, presented. It was very difficult to keep back the intestines by compression, therefore a rapid search was made for the obstruction. It was seen that the whole jejunum and partially the ileum were dilated almost to rupture; the surface of the serosa was dull in color and in places covered with a serofibrinous deposit.

The great omentum was fused into a mass, from which two firm strands extended toward the small pelvis. Several small intestine loops were incarcerated behind the great omentum. The strands were parted, the incarcerated loops freed, and with great difficulty the abdominal wound was closed. Patient, in spite of stimulation, sank after the operation, and died at 3 P.M., July 5th.

Post-mortem, July 6th, nineteen hours after death. Post-mortem record: Body 152 cm. long, of medium-bone structure, musculature, and panniculus adiposus. The general surface is pale. Posteriorly there are pale livories. Rigor mortis is present. Hair of the head is black. Pupils are moderately dilated and unequal. The visible mucous membranes are pale. The neck and chest are well proportioned. The mammæ are of medium size.

The abdomen is slightly distended. In the middle line, beginning 1 cm. above the navel, there is a wound, 21 cm. long, united by means of button sutures.

The external genitalia are negative.

The soft skull coverings, the skull, meninges, and the brain show no pathological changes.

The diaphragm reaches to the third rib on the right side and to the fourth on the left.

The mucosa of the organs of the neck is discolored. The thyroid gland is of medium size and is negative.

The right lung is fixed slightly. The left is free. The lungs show a dilatation of the alveoli along their margins and are congested in general. In the peribronchial lymphatic glands there are isolated caseous nodules.

In the pericardium there is clear serum. The heart is of normal size, pale and soft. The valves and the intima of the aorta are soft.

The mucosa of the trachea is somewhat reddened, and that of the œsophagus discolored by bile.

In the abdomen there is meteorism of the intestinal loops and reddening of the peritoneum. The omentum reaches over the right half of the abdomen and is adherent in the neighborhood of the symphysis to the abdominal wall. To the left of the symphysis there are remains of adhesions. The left portion of the great omentum lies short and free in the neighborhood of the left flexure of the colon. The small intestines are adherent in part to the abdominal wall, but especially in the small pelvis. The liver is small and pale. The spleen also. The kidneys are pale, but otherwise normal. Their pelves are somewhat dilated.

The urinary bladder is somewhat dilated; otherwise normal. The vagina is negative.

The uterus is visibly enlarged, measuring 7 cm. in length, 4 cm. in breadth at the fundus, and 3 cm. in thickness. The walls are 2 cm. thick in places. Musculature is firm. The uterus is drawn backward by adhesions to the sigmoid flexure. On closer examination of the adnexa of the uterus there is seen behind the uterus a cavity, half the size of a man's fist, isolated and filled with pus, in the anterior wall of which, in numerous adhesions, the tubes and ovaries are enclosed. The pus cavity is 6 cm. broad and 3 cm. deep. The tubes are dilated, their walls thickened, and contain much pus. The ovaries are much enlarged, partly cystic degenerated, and partly pus infiltrated.

In the rectum, corresponding about to the reflection of the peritoneum, on the anterior wall, is a 1½ cm. long, reddened, somewhat raised, and irregular ulcer. From this yellow pus flows. This ulcer lies behind the uterus 3½ cm. above the base of the cul-de-sac of Douglas and 13 cm. above the anus. It perforates the rectum from the above-mentioned pus cavity and is seen on the inner wall of the rectum as a pinpoint opening.

The mucosa of the stomach and intestines is pale. Pancreas and the suprarenals are normal.

Bacteriology. Pus from the tubes and the pus cavity shows numerous cocci only, not stained by Gram's method, partly intracellular, having the form of semmelcocci.

Pathological-anatomical Diagnosis. Salpingitis suppurativa gonorrhoica bilateralis; pelveoperitonitis suppurativa ulcerosa cum perforatione recti; adhesiones omenti majoris ad parietem anteriorem abdominis in regione symphysis; status postlaparot; miam propter incarcarationem intestini ilei infimi e compressione adhesionibus omenti effecta; peritonitis diffusa incipiens; tuberculosa obsolata glandularum lymphaticarum peribronchialum.

The second case had the following clinical history: K. O., aged forty-six years, wife of a laborer, was admitted to the Prager Israelitischen Allgemeinen Krankenhaus on May 19, 1904.

Anamnesis. Patient complained of severe pain in the abdomen and of dyspnœa. She claimed to have been always well. Her present illness began about five weeks ago. Without any apparent cause she became ill, complaining of pain in the abdomen, vomiting, eructation, and constipation. Added to the pain in the abdomen, there soon appeared dyspnœa, swelling of the body and feet, which made it necessary to put her to bed.

Patient was pale, sat in bed, dyspnœic, pulse irregular, unequal, rapid, and about 100 to the minute. Temperature, 38° C. Heart action arrhythmic and tones muffled. Heart impulse somewhat external to the mammillary line. The lung findings were those of a bronchitis.

Marked œdema of the lower extremities. Abdomen showed no especial changes. The walls rich in fat.

Palpation everywhere, especially to the left and below, very painful. Liver and spleen not palpable.

Genital Organs. Vagina large. Portio enlarged and directed forward. Body and fundus of the uterus enlarged, moderately firm and retroverted, in the left half quite enlarged and tender. This finding was contemplated as a myoma uteri, and disease of the adnexa was not considered. From the uterus a mucopurulent secretion issued.

Course. The pains in the abdomen lasted unchanged for some time, then moderated, and finally stopped entirely, as also the fever. The further course was that of an uncompensated heart affection, increasing hydrops, dyspnœa, and oliguria. Death due to œdema of the lungs occurred on July 8, 7.30 A.M.

Clinical Diagnosis. Tumor (myoma?) uteri; myodegeneratio cordis; hydrops universalis; hydrops ascites; nephritis interstitialis.

Post-mortem, July 9th, twenty-four hours after death. *Post-mortem record:* Body, 165 cm. long, of strong bone structure and musculature, and rich panniculus adiposus.

Skin is pale. Lower extremities are œdematous. Posteriorly there is hypostatic congestion. Rigor mortis is present.

Hair of the head is black, mixed with gray. The visible mucous membranes are pale. Neck and chest are compressed. Mammæ are rich in adipose tissue, as also the abdominal walls, which are pendulous, œdematous, and show many striæ.

The external genitalia show no pathological changes.

Skull is not opened.

The diaphragm reaches on the right side to the fourth rib, on the left side to the fifth rib.

Thyroid gland is enlarged and contains much colloid.

Mucosa of the organs of the neck is pale.

Both lungs are slightly adherent, and there is in both pleural cavities about half a litre of clear serous fluid. The lungs are œdematous, the right somewhat more dense, and in places in the lower lobes atelectatic.

In the pericardium there is a small amount of clear serum. The heart is dilated, especially in its left ventricle. The heart muscle is pale, yellowish, on the right side somewhat spotted. On the left side the papillary muscles of the mitral valves are grayish on section. The musculature of the left ventricle shows many whitish and dark spots. The valves of the left heart show only slight spots of thickening. The aorta shows great unevenness of the surface and many so-called atheromatous ulcers. The openings of the coronary arteries are free; still, the left shows some irregular thickenings in its course. Between the trabeculæ of the left ventricle some pale thrombi are seen.

The trachea and œsophagus are pale. In the free abdominal cavity there are no abnormal contents. The liver is large, firm, and congested. Also the spleen and the kidneys. The surface of the kidneys is scarred. The pelvis of the left kidney is somewhat dilated, and in it there is some urinary gravel.

The mucosa of the pelvis of the left kidney, as also that of the dilated left ureter, is reddened. At the opening of the left ureter into the bladder there is a protuberance of the mucous membrane of the bladder, caused by a dilatation of the lower end of the ureter, the size of a hazel-nut. On the top of this protuberance can be seen the strictured pinpoint opening of the ureter. The bladder is dilated, and the mucous membrane is somewhat reddened. The vagina is normal.

The uterus is enlarged, measuring 9 cm. in length and 4 cm. in thickness at the fundus. Musculature is firm and in places 2 cm. thick. Between the uterus and the rectum is a cavity, half the size of a man's fist, walled off and filled with pus, measuring 6 cm. in depth. The vermiform appendix is also adherent to its upper wall. The tubes are moderately enlarged and filled with pus. The left tube is perforated into the above-mentioned cavity, 1 cm. to the left of the uterus. The ovaries are included in the adhesions and also are partly infiltrated with pus. The anterior wall of the rectum, to the left of the median line, 4 cm. above the base of the pouch of Douglas, and 13 cm. above the anus, is perforated from the pus cavity by a radiating, sharply defined ulcer, measuring 5 mm. in diameter. Corresponding to the base of the ulcer, on the inner surface of the rectum, there is a perforation the size of a pin-head.

The mucosa of the stomach and of the intestines is pale. Pancreas and the adrenals are normal.

Bacteriological examination of the pus from the small pelvis in cover-glass preparations shows, besides many other small cocci, also many diplococci not stained by Gram's method, partly intracellular, in shape semmelcocci, thus gonococci.

Pathological-anatomical Diagnosis. Endaortitis chronica deformans; myomalacia cordis multiplex; degeneratio adiposa myocardii; dilatatio cordis totius; vegetationes globulosee in ventriculo cordis sinistro; salpingitis suppurativa gonorrhoica bilateralis, subsequenta pelveoperitonitide ulcerosa circumscripte cum perforatione in rectum; calculosis renis sinistri; dilatatio extremitatis inferioris ureteris sinistri e stenosi ostii ureteris vesicalis; dilatatio ureteris et pelvis sinistri cum ureteritide et pyelitide sinistra.

The first case showed a pure gonorrhœal infection. The second was a mixed infection, which might, however, have occurred by entrance of bacteria from the rectal contents after perforation into the rectum, and the ulceration primarily being probably due to the gonococcus only, as is believed by the writer.

Microscopic examination of both cases showed intense infiltration of the tissues of the tubes, uterus, ovaries, and rectum. Both macroscopically and microscopically, the ulceration was seen to have occurred in the external wall of the rectum and then extended crater-like form into the rectum. Staining for bacteria in the tissues revealed the presence of bacilli, cocci other than gonococci, and a few gonococci in both cases. However, the tissues were examined some time after the post-mortem was performed, and thus, as stated above, gonococci could be found only sparingly and mostly in involution forms, which are difficult or impossible to demonstrate, owing to the fact that they do not stain well, or not at all. The presence of other bacteria may be explained by their entrance into the tissues from the rectum after its perforation. That the ulceration occurred from the outside and not the inside of the rectum is positive by the crater-like form of the ulcer in the wall of the rectum extending from its outer wall inward.

In conclusion it may be stated that the gonococcus can produce ulceration in tissues. For demonstration of this process it is very essential that fresh tissues be examined, and these, if fresh, may be stained with any color medium, even with a simple methylene-blue solution, in order to bring forth the gonococci. A pure culture of gonococci may or may not be found in such cases; but the presence of other bacteria in the pus and in the tissues, after perforation has occurred, may be considered as secondary, provided the original process had been due to the gonococcus alone, and the ulceration, both macroscopically and microscopically, points to a process originating from and not toward the side where the gonorrhœal process primarily existed.

Bibliography.

1. Wertheim. Ein Beiträge zur Kenntniss der Gonokokken beim Weibe, Wien. klin. Woch., 1890, p. 25.

2. Frisch. Ueber Gonorrhœa rectalis, Jahresbericht über pathogene Organismen, 1891, p. 117; also Verhandlungen der physikalisch-medicinischen Gesellschaft zu Würzburg, N. F., Bd. xxv., No. 6.

3. Kraus. Nachweis von Gonokokken in den tiefen Schichten der Tuben und Tubenwand, Monatsschrift f. Geburtshilfe und Gynäkologie, 1902, Bd. xvi. p. 192.

4. Wertheim. Zeitschrift f. Geburtshilfe und Gynäkologie, 1896, Bd. xxxv. p. 6.

5. Kiefer. Ibid.

6. Menge. Discussion über Metritis und Endometritis, Centralbl. f. Gynäkologie, No. 45, p. 1145.

7. Jullien. Note sur l'ulceration blennorrhagica, III. Congress international de. dermatologie et de syphilologie à Londres, Journ. de mal. cutan. et syphil., No. 12, p. 741.

8. Kiefer. Loc. cit.

9. Cohn. Monatsschrift f. Gynäkologie und Geburtshilfe, Bd. ii., Heft 4.

10. Kiefer. Loc. cit.

THE DIETETIC USE OF PREDIGESTED LEGUME FLOUR, PARTICULARLY IN ATROPHIC INFANTS:

WITH A STUDY OF ABSORPTION AND METABOLISM.[1]

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AND

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Beans and other legumes have throughout many years past been occasionally recommended as useful in dieting infants, and in Germany especially they are said to be a constituent of a number of preparations of "Kindermehl." So far as we know, however, they have been given in a form in which it is impossible for even adults to take considerable amounts without tending to cause more or less digestive disturbance, particularly if they are given for some time; and statements concerning their effects in infants have been based almost entirely upon somewhat loose and casual observations of clinicians or upon the so-called experience of the laity.

The work that we are reporting was carried out partly in order to determine the possibility, in infants that have persistent difficulty in digesting milk proteids, of administering a useful quantity of vegetable proteid in a form suited to their digestion. We also investigated the question whether this proteid in its influence upon metabolism shows any important differences from milk proteids that are dependent upon the character of the proteid as distinguished from its digestibility and from the mere fact that it is proteid food.

Our results have shown the feasibility of successfully administering vegetable proteid to infants in considerable amounts and for at least a fairly long period. They also suggest that the favorable influence upon nutrition observed in a large proportion of cases is more largely referable to the character of the proteid that we used than to the amount, though this is a point that necessarily has in it an element of undemonstrable theory, and that can be provisionally accepted only after further control studies.

The investigation of the use of vegetable proteid in infants was undertaken at the suggestion of Dr. J. P. Crozer Griffith. The coincident consideration of the probable dependence of the nutritional improvement that occurred in a large proportion of the cases studied, upon the character of the proteid, we based upon the following theoretical grounds: Native food proteids are of various kinds, the chief among them being albumin, nucleoalbumin, nucleoproteid, and a

[1] Read by invitation before the Buffalo Academy of Medicine, December 13, 1904.

less important group most satisfactorily called albuminoids. Albumins, represented for example by egg-albumen, are distinguished by a series of reactions, but, for our purposes, particularly by the fact that they do not contain phosphorus compounds. Nucleoalbumins, conspicuously represented by casein, are especially distinguished by the fact that they contain phosphorus compounds, but do not yield any xanthin bases upon disintegration. Nucleoproteids, represented among animal foods most strikingly by various organs rich in cells, such as the pancreas and the thymus, but found also widespread in the vegetable kingdom, are distinguished chiefly by the fact that they contain a large amount of phosphorus compounds and do yield xanthin bases upon their disintegration. The albuminoids are represented by gelatins and a series of other substances which are of less specialized form than the substances mentioned above.

In human or other animal tissues the protoplasm of the cells is made up largely of albumin, in some cases of nucleoalbumins also; while the nuclei of the cells are formed chiefly of nucleoproteid. Adults receive in their food, in addition to albumin and nucleoalbumin, considerable quantities of nucleoproteid. It is not known how they manufacture the nucleoproteid of their tissues, but it is at least not improbable that the nucleoproteid of the food, or at any rate the nucleic acid that this nucleoproteid contains, is used for a part or the whole of this process. This would at any rate appear to be an economical method as compared with the wholly synthetic manufacture of nucleoproteid from relatively simple substances. In infants, however, the cell nucleoproteid must be manufactured, for the food contains no true nuclein, the proteids of the milk being entirely albumins and nucleoalbumins. If, then, the adult uses his nucleoproteid more or less directly in cell construction, the infant has in this point greater complexity in its metabolism than the adult; and it is possible that this is one reason that the assimilative powers of an infant are so much more subject to uncontrollable breakdown than those of an adult. It seemed possible, therefore, that when the assimilative functions are already incompetent it might be feasible to help the child by giving it nuclein in a form that would be readily absorbed without disturbing the digestion. The difficulties in the way of using animal foods for this purpose in noteworthy amounts are too great, but vegetables of the class of the legumes (beans, peas, lentils) contain extremely large amounts of nitrogenous substances and among these there is a considerable amount of nucleins. That starch can be given to infants in large amounts if predigested is known, and this fact has been used in a special manner by Keller, for example, with apparently very successful results. There was, therefore, no evident obstacle in the way of the use of legume flour except the possibility that the proteid that it contains might disturb the digestion. Heating, however, causes much of this proteid

to go into solution if it is finely ground, and, from a mechanical standpoint, the remainder would appear to be at least no more disturbing than the easily coagulated milk proteid. The practical effects of its use remained to be determined.

In studying this question we have given predigested bean flour to a series of infants, having first convinced ourselves, by the administration of small amounts of the bean flour, that it did not disturb digestion, and having also first studied its absorption and determined that this was, in the infants investigated, satisfactory.

We will discuss first the method of preparing the flour and the manner in which it was used, and then refer briefly to the studies of absorption and metabolism, subsequently discussing the clinical results.

We were unable to find on the market any satisfactory bean flour or pea flour. We, therefore, secured white kidney beans, dried them, and in our first experiments ground a few pounds of flour ourselves in an ordinary spice mill, removing the shell, and using only very fine flour secured by bolting; subsequently we had about two hundred pounds ground for us. It is essential that the flour should be finely ground in order that it may be subject to satisfactory predigestion, and also in order that the proteid may be properly absorbed. There has been much discussion of the question whether vegetable proteid is well absorbed or not (in adults). It seems probable that the correct answer to this question is that unfavorable results are due to the form in which the vegetable proteid is administered. The studies have usually been made with vegetables cooked in the ordinary manner, and in this form the proteid is enclosed in large part in a tough mass of cellulose. Rockwood has shown that, if administered free from cellulose, vegetable proteid is well absorbed. It is, therefore, important to see that the flour is finely ground. The object of predigestion was, of course, to convert the starch, the method of preparation having no effect upon the proteid excepting that due to the heat. In our work we usually prepare a 10 per cent. solution of predigested bean flour, first mixing the weighed or measured bean flour with water until made into a smooth paste, then adding more water until it can be easily stirred, and then heating in a double boiler at a good heat for fifteen or twenty minutes, stirring very frequently. During this time the flour of course swells and a 10 per cent. mixture of flour and water becomes a rather thick paste. It is then cooled to between 60° and 70° C., and the ferment is added and allowed to act for about ten minutes, stirring the mass frequently and loosening the portions that have adhered closely to the sides of the vessel so that they may undergo thorough digestion. Within a few moments after adding an active diastatic ferment the mass becomes perfectly fluid and resembles a rather thin soup. After this, in order to stop the action of the ferment, it is brought to the boiling point; it is then

cooled and is ready for use. The ferment that is to be used is largely a matter of choice, the only essential being that the ferment shall be active and in convenient form. We generally used cereo, sometimes maltine. Any of the other good diastatic ferments on the market would probably do as well. We have used the first mentioned chiefly because it is in very convenient form for this individual purpose and is very active. The nature of the mixture after the digestive process is past has not been investigated farther than to determine that there is little, if any, iodine-reacting starch present and that practically all the flour is in a fluid form. The proteid present would seem from the physical standpoint to be in a condition very suitable for absorption, for it is largely in solution, the solid particles that remain are few and extremely small, and it is impossible to bring down any coagulum by heating, while even strong acids precipitate the proteid only in the form of fine flakes.

In using this preparation with infants we have done as follows: A quantity of the infant's milk mixture approximately equal in food value to the amount of bean flour that we were about to add was discarded and was replaced by 10 per cent. solution of bean flour. For example, with a 3.6.1 milk mixture, the 10 per cent. bean flour is somewhat less than a direct equivalent of an equal quantity of the milk mixture; therefore, in such a case we gave the food as follows: if the child was receiving 48 oz. of 3.6.1. per day this was reduced to 36 oz., and 15 oz. of 10 per cent. bean-flour solution was added; the child therefore got 51 oz. a day, but received about the same energy value (actually slightly less) in its food. In a good deal of the work bean flour was not added at one time to the total quantity of the day's food, but was kept separate and a proper amount was added to each feeding. For example, with previous feedings of 4 oz. of 3.6.1. mixture the child was now given 3 oz. of 3.6.1. and 1¼ oz. of 10 per cent. bean-flour solution. In almost all our work with infants the feedings contained 2.5 per cent. to 3 per cent., at most, of bean flour. From the determinations that we made of the nitrogen in the flour, which will be mentioned immediately, we found that 3 per cent. of bean flour meant about 0.65 per cent. of proteid.

We made, in all, five analyses of the nitrogen in the bean flour. They resulted as follows: I., 3.447 per cent.; II., 3.448 per cent.; III., 3.394 per cent.; IV., 3.559 per cent.; V., 3.327 per cent.; the average being 3.435 per cent. of nitrogen, or 21.468 per cent. of proteid.

In our absorption work we used diapers made of rubber tissue, large wads of absorbent cotton being placed over the genitals to avoid any mixture of the urine with the feces. By this method we could collect the feces on the rubber diapers with entire success and without loss. The only difficulty that we had was that in one instance, when the observations were continued for ten days, the child had rather marked irritation of the skin about the buttocks.

In the one instance in which we did a complete nitrogen metabolism experiment in an infant, we collected the urine by introducing the penis into a piece of rubber tubing, strapping the tubing on with adhesive plaster, the lower end of the tubing discharging into a vessel which collected the urine. In the metabolism experiment in an adult we followed the customary methods. In the case of the infants the nitrogen in the food was determined each day. In the experiment with the adult we used a diet containing substances of which we had many times estimated the nitrogen-content ourselves, or which have a well-known and practically constant nitrogen-content.

The results of our absorption experiments in infants were as follows: Case I. was a boy, aged six months when studied, who had been in the house three weeks, and in this time had gained and lost repeatedly an ounce or two at a time. When the study was begun he weighed six pounds fifteen ounces. He had usually three soft bowel movements daily that were fairly satisfactory. In the preliminary period of study the child received 4 oz. of 4.7.2. mixture every three hours. This period of study was three days in length. The child was then put on 3 oz. of 4.7.2. mixture and 1½ oz. of 10 per cent. bean-flour solution every three hours, and the absorption was studied for a period of six days following. In the two periods the conditions of nitrogen absorption were as follows:

Preliminary Period.		Bean-flour Period.	
Received in three days	8.601 gm.	Received in six days	19.111 gm.
Average daily	2.867 "	Average daily	3.185 "
Excreted in feces in three days	0.503 "	Excreted in feces in six days	1.203 "
Average daily	0.167 "	Average daily	0.200 "
Absorption in three days	8.098 "	Absorption in six days	17.908 "
Average daily	2.699 "	Average daily	2.984 "
Absorption of the intake	94.2 %	Absorption of the intake	93.7 %

The second case was studied at the same time and in the same way. The same amounts of food were given in both periods, but the child vomited about two ounces after one feeding; therefore, the intake in the preliminary period was slightly less than in the first case. This child was four months old when studied; had been in the house six weeks, and had varied between six pounds thirteen and one-half ounces and seven pounds three ounces, and showed the latter weight when the bean flour was begun. There were three or four bowel movements daily, which were yellow, quite well digested, but contained a little mucus. The conditions of nitrogen absorption in this case were as follows:

Preliminary Period.		Bean-flour Period.	
Received in three days	8.422 gm.	Received in six days	19.111 gm.
Average daily	2.807 "	Average daily	3.185 "
Excreted in feces in three days	0.415 "	Excreted in feces in six days	0.548 "
Average daily	0.138 "	Average daily	0.091 "
Absorption in three days	8.007 "	Absorption in six days	18.563 "
Average daily	2.667 "	Average daily	3.093 "
Absorption of the intake	95.1 %	Absorption of the intake	97.2 %

In these two cases the absorption was very good in both periods. In the first case there was only a fraction of 1 per cent. difference in the two periods; in the second case absorption during the bean-flour period was better than during the preliminary period. These figures show that in these children the bean proteid was fully as well absorbed as the milk proteid, and both were extremely well absorbed.

In another child we investigated both absorption and metabolism while bean flour was being given. We had no opportunity to study a control period when the child was taking only milk. The figures for the period of investigation, which was five days in length, are as follows:

Received of nitrogen in five days	14.430 gm.
Average daily	2.886 "
Excreted in feces in five days	1.844 "
Average daily	0.368 "
Absorption in five days	12.586 "
Average daily	2.517 "
Absorption of the intake was therefore	87.92 %

This child's urine was studied daily, with the following results:

First day	0.3192 gm. nitrogen.
Second "	0.2240 " "
Third "	0.1092 " "
Fourth "	0.1041 " "
Fifth "	0.3032 " "
Total in five days	1.0597 " "
Average in one day	0.2119 " "

The nitrogen in the food was	14.4305 gm.
" " feces "	1.8441 "
" " urine "	1.0597 "
Total loss through excretions was	2.903 "
Total retention was	11.527 "
The retention of the intake was	79.87 %

Whether these figures relating to the child's metabolism are wholly reliable or not cannot be said. Some studies of infants in the first few weeks of life have shown extremely high grades of nitrogen retention as compared with those that are observed in adults, even exceeding the retention shown by our figures in this case. This is natural because of the very rapid growth of their tissues at that period, but 79.8 per cent. of retention is so astonishingly high for an infant of the age of our subject that we cannot help suspecting that some of the child's urine was lost. If it was, we could not discover it, a careful watch having been kept on the apparatus regularly without any evidence of loss of urine at any time during the experiment. We do not feel, however, that the figures for metabolism can be further commented upon than to say that if they are correct they show most unusually favorable conditions, particularly for an atrophic infant. The figures for absorption are much poorer than those in the other children and

are not very satisfactory. This was expected in this child, as he came in with a good deal of digestive disturbance, with about five movements daily, which were greenish and contained many curds. The bowels improved after his admission, but this study was begun soon after he came in, when there was still some digestive disturbance.

The further study that we made of metabolism and absorption was carried out in an adult patient, a subject of phthisis. This man had been on a very free diet, having had three meals a day and as large a quantity of milk and raw eggs in addition as he cared to take. He had been gaining quite rapidly in weight on this diet, and his general condition had improved. Because his previous diet had been a very heavy one, we put him, in both the preliminary and bean-flour periods, upon a very free diet as to quantity. In the preliminary period this consisted of milk 2400 c.c.; rice, 30 gm.; eggs, eight daily; bread, 300 gm.; butter, 60 gm.; sugar, 30 gm.; steak, 200 gm.; baked potato, 70 gm. In the bean-flour period the diet remained the same, excepting that the milk was reduced by 550 c.c. each day and in its place he was given each day a solution of predigested bean flour which contained 100 gm. of (dry) bean flour. The nitrogen in the food was estimated by weighing accurately each portion of the food and calculating the nitrogen from the weights. We have ourselves made a large number of nitrogen estimations of the bread, milk, butter, and steak used in the hospital in previous metabolism experiments that we have carried out, and we took the average of these figures for these foods. The nitrogen of the eggs and rice, which is very constant for a given weight, we took from the tables of König and Blyth. The nitrogen excreted in the urine was estimated daily. That of the feces was estimated in the total amount after the feces had been collected throughout the proper period, dried on the water bath (sulphuric acid having been added) and then ground fine. One detail of the work on the feces may be of some interest to investigators in this line. We have repeatedly had the experience, as we had in this instance, of finding that feces contained so much fat that it was impossible to grind them fine after drying them, or to get a homogeneous mixture by occasional stirring while they were drying. We have in some instances overcome this by extracting with alcohol and ether, then drying and grinding the residue and making nitrogen (and when desired, fat) estimations of both the residue and the alcohol and ether extracts. This burdensome procedure, however, proved unnecessary in this instance, and very good results in control estimations were obtained by first extracting with alcohol and ether, then grinding the residue fine after drying it, and then adding the alcohol and ether extracts again to the ground residue and evaporating as quickly as possible, with constant stirring so as to keep the mass thoroughly mixed. In this way a homogeneous mixture was

obtained, and it could also be ground in a mortar much finer than it could before. Control estimations of the nitrogen corresponded very closely.

The results in this case were as follows:

PRELIMINARY PERIOD.

	Nitrogen in the Food.	Nitrogen in the Urine.
October 31	33.0347 gm.	25.4710 gm.
November 1	33.7943 "	23.1336 "
" 2	32.7100 "	22.1450 "
" 3	33.1116 "	21.0112 "
" 4	32.2051 "	25.5780 "
" 5	33.1345 "	18.8160 "
" 6	32.1041 "	21.1232 "
Total	230.0043 "	157 2780 "
Average	32.8577 "	22.4682 "

The total nitrogen in the feces in this period was 12.1866 gm.; the average daily excretion in the feces was 1.7409 gm. The combined urinary and fecal nitrogen in the whole period was 169.4646 gm.; and the daily average excretion was, therefore, 24.2092 gm. Since then the daily average in the food was 32.8577 gm., the average daily retention was 26.3 per cent. of the intake. The absorption of nitrogen was 93.7 per cent. of the intake.

BEAN-FLOUR PERIOD.

	Nitrogen in the Food.	Nitrogen in the Urine.
November 7	30.3704 gm.	22.3264 gm.
" 8	27.5247 "	17.1888 "
" 9	28.6844 "	23.7852 "
" 10	30.7284 "	21.7140 "
" 11	32.4540 "	18.2864 "
" 12	33.2469 "	22.9600 "
" 13	33.9411 "	17.3600 "
" 14	33.2166 "	29.9880 "
" 15	32.9460 "	21.7728 "
Total	283.1125 "	195.2796 "
Average	31.4569 "	21.6977 "

The nitrogen in the feces in this period was 17.0581 gm.; the daily average was 1.8953 gm. The combined urinary and fecal nitrogen was 212.3377 gm.; the daily average excretion was 23.5937 gm. The daily average in the food was 31.4569 gm. Hence, the average daily retention was 7.8639 gm. and the total retention was 70.7748 gm., which was 24.9 per cent. of the intake. The absorption in this period was 94.0 per cent. of the intake.

These figures for metabolism are, therefore, somewhat against the bean flour, since the retention, which was high in both periods, was about 1.5 per cent. better in the preliminary period than in the bean-flour period. The conditions in the early part of this experiment were, however, not satisfactory, for we met the same difficulty that has been noted many times before in attempts to administer legumes as ordinarily cooked, over a considerable period con-

tinuously; that is, after the first few portions, the man objected so greatly to the taste that it was almost impossible for him to take the beans. He made a willing effort to do so, however, and succeeded; but it upset his appetite so much that he was unable to take the proper amount of the other food, as is readily seen by referring to the table for the food nitrogen in the bean-flour period. These figures drop decidedly on the first day, and much more in the next two days; rising again on the next day, and reaching the proper point on the fifth day. The explanation of these figures lies in the point noted; in the first three days it was impossible by any of the means that we used to flavor the beans so as to make them agreeable. The rise in food nitrogen on the fifth day was coincident with the use of cinnamon as a flavor; this was agreeable to the man, and subsequently throughout the period of investigation and a long period afterward he took the predigested bean flour in his milk with entire satisfaction. The last five days of the experiment, therefore, offer a fairer comparison with the preliminary period than does the whole bean-flour period. The figures for these five days are as follows:

Average daily food nitrogen	33.1609 gm.
Average daily urinary nitrogen	22.0534 "
" " fecal "	1.8953 "
Total excretion daily	23.9487 "
Average retention (27.4 % of the intake) was	9.2122 "
Absorption of the intake was	94.2 %

The figures in this part of the bean-flour period are, therefore, 1 per cent. better as regards nitrogen retention than in the preliminary period, and almost 3 per cent. better than in the total bean-flour period. The figures for absorption in this period are very slightly below those in the preliminary period, but the figures for absorption in all three of these periods are so similar that they provide sufficient evidence that this man absorbed the bean nitrogen practically as well as the nitrogen from the milk, eggs, steak, and bread. All were absorbed very satisfactorily, particularly considering the very large food intake.

We cannot draw very definite conclusions from this experiment excepting to say that the bean proteid was quite as satisfactory as the other proteids in its influence upon metabolism. In the last part of the bean-flour period it seemed to have a slightly better effect upon metabolism than the other proteids, though the difference is so slight that little weight can be laid upon it. We could not expect much difference, however, even if nuclein has a noteworthy influence upon proteid metabolism as compared with the influence of other forms of proteid, for this man was already getting some animal nuclein in his food. The experiment serves chiefly to demonstrate the entire metabolic suitability of a large quantity of

legumes in maintaining a large nitrogen retention, and also the satisfactory absorption of the proteid in the legume. The last result directly supports the view previously referred to concerning the absorption of vegetable proteid, and the results with metabolism also support the observations that have been made in considerable number, in adults, in regard to the influence of vegetable diet upon nitrogen metabolism, most such experiments having demonstrated the ready possibility of maintaining a nitrogen equilibrium and of sometimes producing a nitrogen retention, on a vegetable diet. This was usually done, however, by using vegetables cooked in the ordinary manner. The preparation that we used has the advantage of being an extremely concentrated food containing a very high energy value for its bulk and it is at the same time in fluid form. It is, therefore, in these points an excellent food for improving the nutritive condition when it is impossible to take a large bulk or to take solid or semisolid food in considerable quantities. The chief difficulty with it, and one that has as yet been hard for us to overcome, is the flavor, which is distasteful to most adults and older children from the beginning, or soon becomes so. Infants take it without trouble in practically all cases after one or two feedings, but it is difficult to persuade older children and adults to do so. Attempts to accomplish this become largely a question of flavoring, but when flavoring ingredients are necessary over a long period of time a great variety must be used or they too become distasteful. This limitation of the usefulness of this food in older persons may, therefore, remain permanent. The point cannot now be settled.

In infants, however, our clinical results up to the present have been such as to encourage us decidedly. We have not as yet used it in a very large series of cases, as our attention has been chiefly directed to the determination of its influence upon absorption and metabolism, and during this particular period of the year we have not had a large number of cases available for study. Those that have been studied are the following: nine in the service of Dr. Griffith at the University Hospital; five at the Seashore House at Atlantic City studied through the kindness of Dr. William H. Bennett; one private case of Dr. Charles H. Schoff. In these cases the gentlemen who had charge of the cases, or their assistants, usually the latter, prescribed the diet, excepting only that we suggested the amount of bean flour to be used. Another series, that could be studied for only a brief period, was seen at St. Christopher's Hospital. The results in the first fourteen cases, briefly abstracted, are as follows, first mentioning the least favorable cases:

CASE I.—J. P., aged five months; weight ten pounds nine ounces. Treated at Children's Seashore House, Atlantic City. Lost weight for ten days continuously, but slowly. Then weighed ten pounds four ounces. Had been on Meigs' mixture, then on partially peptonized milk. Was put on whey upon the appearance

of decided acute digestive disturbance, in which the stools became green and contained mucus and curds and occasionally a trace of blood. This continued and he was put by one of the residents on three parts of Meigs' mixture and one part of 10 per cent. predigested bean flour, giving three ounces of this mixture every three hours. No noteworthy change occurred in his condition, and after ten days he was taken home, having lost in all one pound from the time of his admission. In our view this case was at this period an unsuitable one for this food or any other except the most dilute.

CASE II.—L. C., a girl aged ten months; weight nine and one-half pounds. Admitted to Children's Ward, University Hospital, with three or four greenish stools daily, containing curds. No acute disturbance. Put on barley-water for twenty-four hours. Then on a 3.6.1. mixture, three parts; 10 per cent. bean-flour solution, one part; five ounces being given every three hours.

The stools rapidly became normal and the child in ten days gained exactly a pound. At this time it developed fever, cyanosis, and rapid respirations. There was no digestive disturbance for five or six days after the onset of this acute illness, when a mild diarrhœa developed and the child one day vomited. The diet was then changed to a plain milk mixture without any noteworthy change in the condition of the digestion; the cyanosis and rapid respiration also continuing. This child died after continued illness. This case is mentioned among the unfavorable ones because one or two of those who saw the child thought that the bean flour was responsible for the acute illness. We personally felt that the attack was not originally a digestive one at all, but was probably due to an obscure bronchopneumonia. Autopsy showed numerous pyæmic abscesses of the lungs originating in middle-ear disease. This case cannot be used for or against the bean flour, though the child temporarily gained very rapidly on the mixture containing bean flour.

CASE III.—J. O., aged three months; weight eight pounds one ounce. Admitted with greenish stools containing curds; five movements daily. This child was the one mentioned as having been studied as to both absorption and metabolism. After one day of barley-water, the child was put on three parts of 4.6.2., one part of 10 per cent. bean-flour solution; why the diet was increased suddenly to this point is not recorded. In the next six days the child gained six ounces; it had never gained as much before. The bowel movements after the first two or three days became soft, yellow, and contained no curds, but there were three or four movements a day. In the following nine days he gained but one ounce more and the bowels were the same. On October 4th he had not gained further and the mother took him home. A more gradual increase in the food might have been more successful.

The others of the cases that were treated at the University Hospital and at Atlantic City are all more or less favorable. We will mention first the University Hospital cases and then the Atlantic City cases.

CASE IV.—Girl, aged four months; weight six pounds thirteen and one-half ounces. This child was the second of our absorption studies. She was admitted having three or four movements a day; they were yellow, half-formed, contained a little mucus. She had lost weight gradually since birth, and had at no time gained; was extremely emaciated and weak. Upon admission she was put on barley-water, followed by Keller's malt-soup; after a week this was changed to a simple milk mixture, containing barley-jelly, which was afterward malted. As the bowels improved, the milk mixture was increased to a 4.7.2., containing malted barley-jelly. Throughout six weeks her weight had varied, with slight rises and falls, between six pounds thirteen ounces and seven pounds three ounces. Bean-flour solution was then added to the 4.7.2. mixture; her weight at this time being seven pounds three ounces. Six days later our bean flour gave out; at this time she weighed eight pounds. She continued gaining after this for four weeks, when she weighed nine and one-half pounds, a simple 4.7.2. mixture having been used during this time. She then lost half a pound in a week, and regained it only very gradually in the next three weeks. This was at the beginning of summer, and her slow gain was thought to be due to hot weather and "hospitalism," and she was sent to the seashore.

CASE V.—The first of our absorption experiments. Was admitted with three to five stools daily that were greenish and loose. He was six months old; weighed six pounds fourteen ounces. He was much emaciated and weak. He was put on barley-water and then on weak milk mixture; gradually increased as the bowels soon became nearly normal to a 4.7.2., which was ordered eleven days after he was admitted. Eighteen days after admission his weight was six pounds fifteen ounces, there having been only slight fluctuation in this period. He was then given bean flour in the 4.7.2. mixture. Six days later, when the flour gave out, he weighed seven pounds fourteen ounces, the bowels remaining the same. Afterward, on a simple 4.7.2. mixture, he gained gradually, in five weeks, to nine and one-half pounds. He was then taken home in satisfactory condition and was lost sight of.

CASE VI.—L. D., aged four months; weight six pounds; very weak. Admitted with mild digestive disturbance; three to five stools daily, which were yellow and partly formed, but contained some curds. Put on barley-water, then on dilute milk mixture, gradually increased. Five weeks after admission was given 4.7.2. mixture. The child was then having two or three movements a day, which were yellow and quite well digested, but there had been

no gain. Six weeks after admission was given bean-flour solution in the 4.7.2. mixture. Four days later she weighed eight and one-half pounds. Was taken home just after this, weighing eight pounds nine and one-half ounces. Readmitted a week later with loose, greenish stools, and having lost half a pound; she had been on a simple milk mixture outside the hospital. Was put on barley-water, then on her previous milk mixture, and gained half a pound in five days. After this stayed stationary for a week and was then given bean-flour solution again, in her milk mixture, and gained a half-pound in four days, and a quarter of a pound in the next five days, and was again taken home in satisfactory condition, weighing nine and one-quarter pounds. The gain in this case when bean-flour solution was first given was almost incomprehensible. At each weighing before this time the record had been within an ounce or two of six pounds. At each weighing after five days of bean-flour solution the record was regularly above eight and one-half pounds. She was carefully weighed by the nurse, the resident physician, and by one of us, all records agreeing. The scales may have been at fault, but they were correct when tested afterward. If the gain was as recorded it was certainly not all due to increase of tissue, for in all her food during this time she received a little less than enough solids to have made this gain if all had been deposited in the form of tissue. Hence, some of it must have been retention of water. Water retention, however, is a very desirable thing in most atrophic infants, as their tissues are usually very abnormally dry. Whatever may be said of the weight records, the improvement in the child's condition was perfectly astonishing; within four days she became a wholly different-looking infant, and she retained her improvement.

Case VII.—Girl, aged thirteen months; weight eleven pounds, one ounce; very ill-nourished and weak. Admitted with five or six greenish stools daily, which contained mucus. Put on barley-water and then on a milk modification which was increased to 4.7.2. mixture, although the stools remained about the same. She gained a half-pound in the first ten days; then remained stationary for a week. Was then put on bean-flour solution. The bowels improved, but the movements still remained greenish and loose for a week, and in this time she lost a half-pound. From this period on, however, the bowels rapidly improved and soon became normal, and in the next three and one-half weeks she gained one and one-half pounds, and was discharged weighing one and one-half pounds more than when admitted, her bowels for two weeks having been entirely normal.

Case VIII.—C. B., girl, aged six months; admitted weighing seven and one-half pounds, with three or four greenish, half-formed movements daily. Put on barley-water for twenty-four hours, followed by simple milk modification. Bowels did not improve,

and the child lost a half-pound in four days. Bean-flour solution was then added. The bowels within three days became normal, and in ten days the child gained slightly over a half-pound; in five days more another half-pound; in three days more another half-pound. Was discharged, with normal bowels, weighing eight and one-half pounds.

CASE IX.—D. L., boy, aged seven months. Admitted weighing nine pounds, with four or five movements daily, which were green and contained mucus and an occasional trace of blood. Put on barley-water, then on milk mixture; the bowels improved at first, but after three days remained stationary, continuing loose and greenish, with some mucus. Nine days after admission the child was on a 4.7.1. mixture; weighed nine and one-half pounds, but had been stationary for nearly a week. Then bean-flour solution was added; three days later the bowels were recorded as nearly normal, the child still weighed nine and one-half pounds. Six days later had gained a half-pound; four days later another half-pound; four days later than this another half-pound. Was discharged in satisfactory condition.

CASE X.—K. D., boy, aged nine months; weight twelve pounds; ill-nourished and weak. Three to five yellowish stools daily, containing curds. Put on barley-water and then on milk mixture, which was increased to 3.6.1. mixture and afterward up to 4.7.2. He had gained three ounces in five days. Wheat flour, predigested as the bean flour had been, was then added. The child gained four ounces in four days; then remained stationary for three days. Then predigested bean flour was substituted, and in the next three days there was a gain of nine ounces; in the next week four ounces; in the next week twelve ounces. Was discharged weighing fourteen pounds. The bowels had improved on the milk mixture and remained in good condition with the use of both wheat flour and bean flour.

The following four cases were treated at Atlantic City, by Dr. Wm. H. Bennett and Dr. Sidney Repplier:

CASE XI.—E. W., girl, aged seven months; weight fourteen pounds four ounces. Stools fairly normal; weight decreased regularly for a week, during which time she lost half a pound. At that time the bowels became loose, contained mucus, and were greenish. She had been on Meigs' mixture. To this was added, through a misunderstanding, over 4 per cent. of bean flour, with the result that vomiting occurred after every feeding. She was then put on peptonized milk and two days afterward was put back on Meigs' mixture containing 2½ per cent. of bean flour. Her weight was then thirteen pounds; stools greenish and contained mucus and curds. One week later had lost four ounces, but the stools were improved. Three days later gained two ounces, stools much improved. One week later gained five ounces, stools normal;

four days later gained five ounces more, stools normal. Was then taken home in good condition.

CASE XII.—Boy, aged five months; weight ten pounds eight ounces. Stools greenish and loose. Lost on Meigs' mixture six ounces in a week. Was then given 2½ per cent. bean flour in Meigs' mixture. Three days later stools much improved, had lost two ounces; three days later stools normal, had gained four ounces. Was then taken home by the mother.

CASE XIII.—A. E., aged seven months; weight twelve pounds; stools greenish, with curds. On Meigs' mixture, from July 1st to 26th, lost a pound. Bowels remained bad. Put, through misunderstanding as in the first case, on excessive amount of bean-flour, getting over 6 per cent. Vomited occasionally for a day. Amount of bean flour reduced to 2½ per cent. Vomiting stopped at once, stools improved rapidly. Three days later stools normal excepting for a little mucus; had gained nine ounces; three days later had gained seven ounces more. Stools normal. Developed chicken-pox and was sent home.

CASE XIV.—E. H., aged six months; weight eleven pounds four ounces; stools normal. Gained four ounces in a week; stools then became loose, and contained a little mucus. Lost six ounces in ten days. Had been on Meigs' mixture. Then bean-flour solution added. Gained four ounces in three days; stools improved. Gained ten ounces in the next five days; stools normal. Discharged in good condition.

In addition to these cases there were treated at St. Christopher's Hospital, in the service of one of us (Edsall), thirteen infants (Cases XV. to XXVII., inclusive), who for a short time got milk mixtures containing predigested wheat flour or predigested bean flour, the wheat flour having been used at first because bean flour could not be secured at that time. Unfortunately bean flour was obtained only a very short time before the term of duty on this service was ended, and hence these observations are of little value. They serve chiefly as a contrast between the effects of wheat flour in these cases and of bean flour in the cases that have already been detailed.

Of the thirteen cases, six were treated with mixtures containing predigested wheat flour, but got no bean flour, receiving the wheat flour in various cases for from ten days to three weeks. Five of these six lost persistently; one gained nicely from five pounds twelve ounces to six pounds twelve ounces in ten days, after having lost for the preceding ten days. The bowels in this case remained slightly loose. Three other infants got wheat flour for from ten days to two weeks, one gaining a few ounces at first and then losing, the others losing persistently. When bean flour was secured all these three were apparently near death, and all ultimately died; but upon the use of bean flour one held its weight for a week and then rapidly lost, while the others gained four ounces each in the first

three days, and then died unexpectedly without any new symptoms. In all these cases the bean flour seemed to be well digested, and in all of them the bowel movements became more satisfactory under its use. Two other infants that had been on predigested wheat flour showed decided improvement of the bowels under bean flour, but were sent to the seashore in three days after its use without any record of their weight at the time. Another child had lost rapidly on predigested wheat flour and showed no gain on bean flour, though it held its weight for a week, when it suddenly died. It seemed *in extremis* when bean flour was started. Another child came in with a mild acute enterocolitis. It got barley-water for forty-eight hours, then whey, afterward whey and albumin-water alternating. The bowels remained persistently bad for a week, and the infant lost a pound. It was then given whey with 1½ per cent. of fat added and 2 per cent. predigested bean flour. The bowels became normal almost at once, and the infant gained a pound and a quarter in three days, and was taken home by its mother and lost sight of.

In none of these cases, excepting the last, was the child in a condition that permitted of any special hope from any treatment at the time that bean-flour solution was started. The results with wheat flour (that was given in almost the same form that Keller gives it in his malt-soup; though it was digested with another ferment) were very unsatisfactory in eleven out of twelve instances. One child, as stated, showed rapid improvement, but the others lost persistently, and no very satisfactory influence upon the bowels was observed; though in no instance did it seem to produce any further digestive disturbance. Hence, the results with wheat flour appear to be much less satisfactory than those with bean flour, though it is, of course, not wholly fair to contrast a group of cases in one institution with those in another. On the whole the St. Christopher's cases were more advanced when the wheat flour was started than were most of the cases at the University Hospital or at Atlantic City that were on bean flour; but in none of the cases at St. Christopher's Hospital was there any very recent acute disturbance of the digestive tract, excepting in the one last described. All the cases, including the last, were atrophic infants, their weights ranging from five and one-half to nine pounds. Three of the infants were under ten weeks of age; one of these was the infant that gained rapidly on wheat flour; the other two did not gain. One other was four months old, and the remainder were between six and eleven months of age.

In addition to atrophy in infants, we have thought that predigested legume flour might be of value in the management of cases in older children or in adults in which there is persistent digestive disturbance and difficulty in taking a sufficient amount of food, particularly proteid food; or in which there is malnutrition from other causes, and the food intake is unsatisfactorily low.

Our observations on this point have been as yet very fragmentary and we do not now feel extremely hopeful in regard to its practical usefulness in most of such cases unless some means of flavoring it satisfactorily can be devised; for, as stated, adults and older children object at once, or after taking a few portions, to the taste of the beans. The man on whom we did the metabolism experiment gained four pounds in the preliminary period (that in which no bean flour was used). In the first four days of the use of bean flour, when his appetite was much upset, and he took a much reduced amount of food, he lost two pounds; but in the next five days he gained three pounds, so that the gain in the first period and the last part of the bean-flour periods was practically the same. Afterward he went back to his usual diet and lost a pound a week for two weeks. He then requested that he be allowed to take predigested bean flour again. Without making any other change in the diet, he did this, and gained one pound each week for two weeks (the bean flour being then flavored), and his appetite improved together with his gain. The conditions in this case are hardly worthy of special comment, though rather favoring the bean flour.

In another case, however, very remarkable results were obtained, and this case is sufficient to encourage the further use of the preparation in children past infancy.

Case XXVIII.—A girl, aged two years, who in brief had the following history: She had been running down for a year; had had persistent digestive disturbance during this time, with constant diarrhœa and occasional vomiting, and nothing seemed to agree with her. She had constantly lost weight at the time when seen by Dr. Charles H. Schoff, of Media, who treated her with predigested bean flour. She had been for three months before this at Atlantic City, under the care of several skilful clinicians, and was then seen by Dr. J. P. Crozer Griffith, who tried a variety of diets without success. The child seemed entirely unable to take milk even when it was greatly diluted, and other liquids agreed but little better. She was then tried on semisolid and solid food, but grew decidedly worse. She was referred to Dr. Schoff at this time, and was then having from ten to fifteen bowel movements a day, which were loose and green. She was vomiting several times daily; was emaciated and extremely weak; her hands and feet were cold, and she was apparently in a desperate state. She weighed eighteen pounds. Several days of careful dieting and medication brought no improvement, and at our suggestion Dr. Schoff tried predigested bean-flour solution, alone, in small amounts. Finding that this did well it was rapidly increased, and the child was given eight ounces of 10 per cent. solution every three hours. Dr. Schoff states that within twenty-four hours the bowel movements were much reduced and much improved in appearance, and the vomit-

ing had stopped. Within a few days more the bowel movements were reduced to about three daily and the child's general condition was greatly improved, and after this there was continuous general advance in health and improvement of the digestive tract. In the first week a gain of slightly less than a pound was made; in the second week a similar gain; in the third week a little over two pounds; in the fourth week two pounds. At this time it was attempted to give the child a very small amount of milk with the bean flour, but it immediately disturbed the digestion. Two weeks later other foods were gradually added, and the child when last heard from had continued to gain and was to all appearances perfectly normal excepting for a tendency to intestinal disturbance from slight causes. This child objected to the taste of the bean flour at first, but took the subsequent feedings readily.

In this case the usefulness of the preparation probably depended upon the fact that it is extremely easy of digestion, has a high nutritive value for its bulk, and contains (that which most other very easily digested preparations do not) an amount of proteid that is entirely sufficient for the needs of the organism. In similar cases, even when much less severe, it would be worthy of trial. It would also be worthy of trial (if agreeably flavored) in cases of acute infectious diseases, particularly in typhoid fever, when there is difficulty in digesting the proper amount of milk. In these cases the addition of a small amount of this preparation would increase the nutritive value of the food, and would sometimes improve the digestion—if in no other way, through its action upon the curd of the milk. It has become the custom of one of us, in a hospital service in which large numbers of cases of typhoid fever are seen, to treat any digestive disturbance on exactly the principles that are used in the treatment of digestive disturbances in infants, reducing the fats or the proteids or both as seems wise, and frequently adding a cereal to prevent the formation of large, tough curds. The latter is a particularly useful procedure in a certain number of cases, and we have seen it rapidly control serious disturbance of digestion, with diarrhœa, distention, etc., in typhoid cases. Predigested bean flour in the form in which we use it has, as we could readily determine, the same influence upon the curd as barley-jelly and the like, and it has also another influence—it is necessary to add much more acid to milk to curdle it when predigested bean flour is present than when it is not. This is probably due to the acid combining with the proteid of the bean.

These clinical results seem to us on the whole to be decidedly favorable. It is to be remembered that all the infants treated were hospital babies, and that with one exception (the last St. Christopher's Hospital case) they had all been losing before the bean flour was started, or had at best merely maintained a very low weight. We would also have it especially noted that no change was

made in the diet excepting that a portion of what the infants had been getting immediately before was cast out, and in its place an equivalent, or usually very slightly less, value of bean flour was given. All the St. Christopher's Hospital cases, excepting the last, are of little importance, since all were in extremely bad condition when the bean flour was started, and only three of them got this preparation as long as a week. These three did a little better than they had done for some time before, though they all died. Of the cases treated elsewhere, three have been classed as unfavorable. The first of these was at the time probably not in condition to receive this or any other food, except the most dilute, and hence was certainly not a suitable case for trial of the food. In the second case there was a rapid gain at first, and then an acute illness which was not due to the diet. In the third case there was some gain, though on the whole the infant did not do well; but the milk mixture used was certainly very concentrated for an infant of the age and digestion of this one, and a lower modification might have done better. In none of these instances was there any actual evidence that the food did harm. The other infants all gained and all were continuously improving when they passed from observation, most of them having increased largely and rapidly in weight and strength and many of them having much improved digestions. Eleven out of fourteen infants, therefore, showed decided improvement, and most of them showed very striking improvement.

Atrophic infants, when treated in hospitals, certainly do not usually show so high a percentage of improvement and such large gains. Indeed, in our experience, and certainly in that of most other observers, such cases are, in hospitals, most unsatisfactory to treat, and usually show persistent downward progress. We think, therefore, that there is good reason to encourage a more extensive trial of the preparation.

There are, of course, several evident reasons why one may be doubtful whether the legume flour had any influence in these cases that was peculiar to itself. The readiest objection that could be raised we have already met—the improvement was certainly not due merely to increase in the amount of food, for the amount of bean flour was purposely kept only equal to or a very little below the amount of milk mixture that it displaced. Those who use very low modifications may consider that some of the infants, at least, had had too strong a milk mixture previously, and that the improvement was due to dilution of the milk. There may be some truth in this; for some of the milk mixture was displaced by the bean-flour solution, and there was, therefore, a little dilution of the milk, but if this is the explanation of the results it means that these babies digested and absorbed the bean-flour solution better than they did modified milk, for the food as a whole was not appreciably diluted, the bean flour, as stated, maintaining

the actual concentration of the total food at practically the previous point. Another possibility that might be suggested is that improvement did not occur because of any influence of bean flour as such, but merely because of its mechanical effect on the curd. This is almost certainly not the case, for most of the babies had already received either barley-water, barley-jelly, malted barley-jelly, predigested wheat flour, or arrowroot.

Three points are, however, subject to somewhat serious criticism. In the first place the number of cases is small, and a larger number may show that our results were due simply to chance good fortune. Again, our cases were necessarily under observation for but short periods, and longer observation is necessary before it can be stated whether infants can often be brought back to good health by this means. Further, we have suggested that the bean flour probably exercised a special influence directly upon metabolism; that is, that it did not act solely through its influence upon digestion, or through the fact that an increased amount of food was being absorbed by babies who had previously been unable to take enough. This we have not definitely proved, but in support of our suggestion we have the following facts: While it is true that in most instances the digestion was more or less decidedly improved under the use of bean-flour solution when there was previously distinct digestive disturbance, it is equally noteworthy that in a number of instances the digestion was but little disturbed beforehand. In most instances also the gain in weight and strength was almost immediate, and it was usually very rapid, while the digestion in repeated instances improved only much more gradually; so that it appeared in such cases that the improvement of digestion was a part of the general improvement rather than the cause of it. Further, and most important, there was in some instances little or no change in digestion, and yet very marked improvement occurred; for example, in the first two cases in which absorption was studied the bowel movements of the two periods remained about the same, and the figures show that the actual absorption was already good and practically did not change when bean flour was given, yet both infants gained nearly a pound in six days, and afterward continued to gain while they had previously been stationary. That this food is capable of causing much improvement of digestion was shown by a number of the cases, most strikingly by Dr. Schoff's very remarkable case in the two-year-old child, in which the result was probably due chiefly to the influence on digestion. Disturbance of digestion, however, does not constitute the whole of the pathology of infancy. In dealing with adults there is an increasing and proper conviction that a good deal more digestive disturbance than was once thought is dependent upon some general disorder of nutrition; that is, that the disorder of digestion is not the primary condition, but is itself a result of some other disease; and the fact that digestive dis-

turbance is of enormous importance in infancy and early childhood need not, as it often does, render obscure the similarly important fact that in not a few instances in infants also the most important disturbance is one of general nutritive processes rather than of the digestive organs.

In order to give satisfactory evidence that the successful results in our cases were due to a special influence of the bean flour, and, particularly, to a special influence of the bean proteid, we must, however, demonstrate that similar results cannot be obtained in a similar proportion of cases by means of predigested starches of other forms. To a limited extent we have given evidence of this in the cases so far studied. One infant had been on Keller's malt-soup, and also on malted barley-jelly, with no improvement; another infant was treated with predigested wheat flour with slight improvement, then became stationary, and immediately afterward gained rapidly, as long as it was under observation, when we used bean flour. The cases seen at St. Christopher's Hospital also favor somewhat the view that wheat flour produces less successful results than bean flour.

If legume flour has this special influence upon the metabolism of atrophic infants, we think it may most readily be thought that it is due to the effect of the nuclein, or possibly to other particular substances in the flour, but not to the mere gross quantity of proteid in it, for the actual amount of proteid absorbed, as seen in the experiments detailed, was almost the same when bean flour was being used and when it was not.

SUMMARY. Bean flour in which the starch is predigested by means of a diastatic ferment seems to be well digested and absorbed by infants and adults. An extremely concentrated food may be given in this way in fluid and partially digested form; a 20 per cent. solution, although fluid, is practically equivalent to beefsteak in nutritive value. Its influence upon the digestive tract in infants in the cases studied was usually distinctly favorable, and its influence upon metabolism in infants and adults is at least equal to that of milk. Of fifteen infants treated, one did not gain; one gained rapidly, but had an intercurrent illness and the flour was stopped; one gained nine ounces and then almost ceased to gain. The others gained as follows: one, fifteen ounces in six days; one, thirteen ounces in six days, both continuing after the bean flour gave out; one, one and a half pounds in sixteen days; one, one and a half pounds in twenty-three days; one, one and a half pounds in seventeen days; one, over two pounds in four days, and after readmission twelve ounces in eleven days; one, one and a half pounds in twenty days; one, twelve ounces in eleven days; one, four ounces in three days (then taken home); one, one pound in seven days; one, one pound in eight days; one, one and one-half pounds in three days. All these were atrophic infants that had previously been stationary or

losing. A child of two years that had had persistent and very dangerous disturbance of digestion with advanced malnutrition improved immediately, the digestive tract became nearly normal within a few days, and the child repeatedly gained over two pounds a week. The last-mentioned child took nothing but bean-flour solution; the infants took usually about 2½ per cent. of bean flour in milk modifications.

These results are certainly unusual. They need to be controlled in several ways before any definite conclusions can be drawn from them, but it seems possible that they were due to a special influence of the legume flour on metabolism, and, perhaps, to a particular influence of the nuclein contained in this flour upon the tissue-building processes.

One point that appears to be of some importance we have definitely determined: it is easily possible to administer in this way as much as 0.75 per cent. to 1.0 per cent. of proteid, a fact of decided consequence in those common cases in which it is difficult or impossible to administer a proper amount of milk proteid.

It is desirable to test this preparation further in older children and adults who are the subjects of malnutrition. This will necessitate, however, some method of preparing the bean-flour solution by which it can be pleasantly flavored, as when unflavored its taste prevents its use with older patients for any considerable period. Infants, however, take it readily in milk.

THREE CASES OF POISONING BY POTASSIUM CYANIDE.

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Cases of poisoning by cyanide of potassium are not frequent in this country, being met with more frequently in England, Germany, and France. The drug is usually taken with suicidal intent; thus, in 402 cases of cyanic poisoning collected by Witthaus[1] in 65.4 per cent. of cases the poison was taken for this purpose. Of this number cyanide of potassium was the drug used in 83 cases. Of the 256 suicides, but 43 were women, and of those who took cyanide of potassium 5 were relatives of photographers and 4 of platers. This fact is of interest, as the majority of cases reported occur among photographers, electroplaters, mirror-makers, chemists, and soldiers, their occupations rendering the procuring of the drug comparatively easy.

I am indebted to Dr. John D. Curran, of Binghamton, New

York, under whose observation the first case came, for permission to report it.

CASE I.—A. B., female, aged thirty-eight years, hair-dresser by occupation. Had been dyeing a patron's hair during the afternoon with a dye containing silver, thus discoloring her fingers. On her way home from work she entered a drug-store, asking for ammonia with which to remove the stains. The clerk gave her several large pieces of cyanide of potassium, telling her that "this is better than ammonia," advising her to moisten her fingers and rub them with the cyanide of potassium, but failing, either through ignorance or carelessness, to inform her of the poisonous properties of the drug. At ten o'clock that evening she entered her bath-room, a small room (dimensions about 6 x 8 feet), imperfectly ventilated by one small window opening into an air shaft. Selecting a piece of cyanide of potassium about the size of a hickory-nut, she rubbed it vigorously on her fingers and hands, removing the stain. This took about five or ten minutes, at the end of which time, *and before she had an opportunity of washing her hands*, she was suddenly attacked with vertigo, felt faint, "everything turned black before her," and she experienced much difficulty in breathing. Her husband, in an adjoining room, heard her scream, and, rushing in, found her unconscious. He carried her into the fresh air and succeeded in a few minutes in restoring her to consciousness, she then taking about two ounces of whiskey. She improved rapidly, and soon was able to walk up stairs and go to bed. At 11.30 P.M. she became much worse, vomited freely, and Dr. Curran was called. He found her with her lips, fingers, and extremities deeply cyanosed. She was in a condition of shock, as evidenced by her subnormal temperature, cold perspiration, rapid, weak heart action, and sighing respiration. Under repeated hypodermic injections of strychnine sulphate, $\frac{1}{30}$ grain, and atropine sulphate, $\frac{1}{100}$ grain, with the application of external heat, she improved for about one and one-half hours, when she became restless, active, and tossed her head from side to side. This condition of motor activity was probably due to the action of the strychnine, which had been given freely during the period of collapse. She was readily quieted by the administration of morphine sulphate, $\frac{1}{8}$ grain, given hypodermically, and fell into a quiet sleep, from which she awoke the next morning weak and somewhat prostrated. During the following day she was nauseated, her heart action rapid and weak, and she continued to give some evidences of shock. She made a rapid and uneventful recovery, being up and about the third day.

The following cases came under my own observation while resident physician in the Pennsylvania Hospital, Philadelphia:

CASE II.—George R., aged thirty-five years, mirror-maker, admitted to the Pennsylvania Hospital February 8, 1901, with the following history:

About fifteen minutes before his admission to the hospital he entered a saloon, sat down, and called for a drink of whiskey. Upon receiving the liquor he placed a small quantity of a white substance into it and a few minutes later picked up his glass and drank the contents. He almost immediately fell forward on his face, unconscious, without uttering a sound. He was hurried to the hospital, where upon admission his condition was as follows: Completely insensible; face cyanosed. Respiration somewhat jerky in character, twenty per minute. Jaws clenched, requiring a gag to open them. Some mucus in mouth. Characteristic odor of bitter almonds on breath. Pulse weak and small. Eyelids closed; eyes fixed; pupils dilated, equal, did not react to light or accommodation; conjunctival reflexes absent. No escape of urine or feces.

Patient was immediately given a hypodermic injection of strychnine sulphate, $\frac{1}{10}$ grain; his jaws separated, the stomach tube introduced, and his stomach thoroughly washed out with warm water. Sylvester's method of artificial respiration was instituted. Despite this the cyanosis increased rapidly, and he died eight minutes after admission. A considerable quantity of cyanide of potassium, properly labelled, was found in his pocket.

The post-mortem examination made by the coroner's physician, Dr. Wadsworth (to whom I am indebted for the findings), showed the heart to be normal, excepting for a considerable deposit of fat; the lungs were congested. The stomach was empty; the mucous membrane much inflamed, especially toward the pyloric end. The liver and kidneys were slightly engorged.

CASE III.—X., male, aged about forty years, was found dead in bed in a hotel. He was last seen alive about eight hours before his body was discovered.

The post-mortem examination made by the coroner's physician, Dr. Morton (who kindly furnished me with the findings of the examination), showed results quite similar to those found in Case II. The heart was normal, excepting for beginning atheroma in the aorta. The lungs were much congested and dark in color. The larynx was also much congested. The mucous membrane of the stomach was greatly inflamed, especially at the pyloric end. The odor of hydrocyanic acid was noticed immediately upon opening the stomach. The liver was engorged, as were also the kidneys. The mucous membrane of the bladder was not inflamed.

Dr. Curran's case (Case I.) is of great interest, as there was apparently poisoning by cyanide of posassium by two methods in the one case. The unconsciousness and vertigo, which the patient first experienced, were unquestionably caused by the inhalation of the drug in a small, poorly ventilated room. The period of shock, occurring an hour and one-half later, was undoubtedly due to the absorption of the drug through the skin, it being recalled

that after removing the silver stains from her hands with the cyanide, the patient had no opportunity of washing her hands, falling unconscious before she could do so. That poisoning by these two methods may, and does, occur is proven by cases reported by Souwers[3] and by a writer in the *British and Foreign Med.-Chir. Review.*[3]

The minimum lethal dose of cyanide of potassium varies, according to the different authorities, but is generally fixed at from 2 to 5 grains, Bennett[4] reporting two cases with fatal results after taking 2 grains and 4⅓ grains, respectively. Death does not occur as rapidly in cases of poisoning by potassium cyanide as in cases of hydrocyanic acid poisoning, usually not taking place for from fifteen minutes to an hour after the ingestion of the drug. Cases are reported, however, in which death has taken place in less than ten minutes, Casper-Liman[5] reporting a case in which the drug was taken with suicidal intent by a young woman twenty years of age, death occurring "immediately." Valcourt[6] and Haskins[7] mention cases in which death resulted in two and five minutes, respectively. The mortality in cases of cyanic poisoning is high, Witthaus[1] stating that in 455 cases, 382, or 84 per cent., died. Death in these cases is due to paralysis of the respiratory centre, although it would appear that in some cases it is caused by the depressant action of the drug upon the heart itself.

That recovery frequently takes place, even after the ingestion of large doses, is shown by cases reported by Higgins,[8] Wiglesworth,[9] Stevenson,[10] Quintin,[11] Brockett,[12] and Gillibrand,[13] in which from 19½ grains to 50 grains of cyanide of potassium have been taken, the patient in each instance recovering. While death takes place more slowly in fatal cases of cyanide of potassium poisoning than in fatal cases of hydrocyanic acid poisoning, recovery is more delayed in these cases than in those of hydrocyanic acid poisoning. Unconsciousness generally persists from two hours to six or eight hours, Dobson,[14] Quintin,[11] and others reporting cases in which the period of unconsciousness extended over this length of time, and one remarkable case is reported[15] in which the unconsciousness persisted for three days, the patient not being discharged from the hospital for ten days. In the other cases cited the patients have usually been discharged in from three to four days.

Autopsy has usually shown the left ventricle of the heart empty and firmly contracted, the right containing uncoagulated blood. The stomach is frequently found much inflamed, especially toward the pyloric end. The lips, mouth, and stomach at times show evidences of corrosive poisoning, probably due to the carbonate of potassium used in the manufacture of the cyanide of potassium.

The most important factor in the treatment of cases of cyanic poisoning is the promptness with which it is instituted. Immediate evacuation of the stomach and intestinal canal, the administration

of cardiac and respiratory stimulants, artificial respiration, friction of the extremities, and cold affusions to the spine, with the patient in a warm bath, or the use of the alternate hot and cold douche to the spine, offer the best results. Various drugs have been suggested as additions to water in washing out the stomach: hydrogen peroxide; potassium permanganate; ferric and ferrous salts in combination; carbonate of potash in solution or in combination with sulphate of iron and ether. Of these probably the best is the permanganate of potassium, the use of which is recommended by Kossa[16] and other writers. Witherstine[17] calls attention to the article of Heim,[18] in which the author states that "morphine seems to be the antidote to cyanide of potassium, and vice versa."

REFERENCES.

1. Witthaus. Toxicology, 1896, vol. iv. p. 604.
2. Souwers. Philadelphia Medical Times, 1877–1878, vol. viii. p. 345.
3. British and Foreign Med.-Chir. Review, July, 1876, p. 231.
4. Bennett. Medical Record, 1894, vol. xlv. p. 742.
5. Casper-Liman. Handbuch d. ger. Med., 8te Aufl., Bd. ii. p. 496.
6. Valcourt. Union médicale, 1875, 3 s., tome xx. p. 626.
7. Haskins. Boston Medical and Surgical Journal, 1870, vol. lxxxii. p. 21.
8. Higgins. Medical Record, 1891, vol. xl. p. 687.
9. Wiglesworth. British Medical Journal, 1897, vol. i. p. 1039.
10. Stevenson. Lancet, 1871, vol. i. p. 806.
11. Quintin. Berl. klin. Wochenschrift, 1885, Bd. xxii. p. 120.
12. Brockett. Lancet, 1886, vol. ii. p. 1174.
13. Gillibrand. Lancet, 1876, vol. ii. p. 223.
14. Dobson. British Medical Journal, 1896, vol. i. p. 17.
15. Med.-Chir. Rundschau, 1867, 195, ex Friedreich's Bl. f. ger. Med., 1868, Bd. xix. p. 452.
16. Kossa. Therap. Monatsh., 1892, Bd. vi. p. 549.
17. Witherstine. Sajous' Cyclopedia of Practical Medicine, 1902, vol. v. p. 337.
18. Heim. Münch. med. Woch., 1896, No. 37.

EVENTRATION OF THE DIAPHRAGM, WITH A REPORT OF A CASE.

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Eventration of the diaphragm has been known ever since J. L. Petit reported and named a case in 1790. It may be described as an abnormally high position of the left half of the diaphragm, with dislocation upward of the abdominal viscera, particularly the stomach, on the left side; hypoplasia of the left lung, and displacement of the heart to the right. It gives rise to physical signs closely resembling those of diaphragmatic hernia. Its proper

classification has been a matter of considerable difficulty. The greater number of writers (Laacher, Thoma, Leichtenstein) include it among the forms of diaphragmatic hernia, regarding the distended diaphragm reaching high into the abdominal cavity as a part of the hernial sac, the other layers being the pleura above and the peritoneum below. Others (Reinhold) consider the chief feature the hypoplasia of the lung. A third group (Crispino) regards such cases as instances of dextrocardia, a fourth group (Doering) as primary defects of the diaphragm, and a fifth group (Cruveilhier, Glaser) as possibly acquired lesions.

The following case was observed at the Philadelphia Hospital in the service of Professor James Tyson, to whom we desire to express our thanks for permission to publish it:

H. H., white, aged twenty years, a farmer.

Family History. His father died at the age of seventy; his mother was living. She was said to be nervous and to have had "spells" until she was twenty years old. Five half-brothers and three half-sisters were all said to be nervous.

Present History. Patient had one convulsion in infancy, a second at the age of fifteen years, and since then they had occurred about once a week; otherwise he had been healthy. The convulsions appear to have been typical epileptic attacks commencing with nausea and vertigo, and followed by unconsciousness, during which the patient fell to the ground, but rarely injured himself. Sometimes he bit his tongue. After the attacks he felt exhausted, had headache, and ordinarily slept for two or three hours. When he awoke he complained of peculiar dreams. He was admitted to the ward for nervous diseases in the service of Dr. Charles W. Burr, where it was noted that he was well developed, well nourished; that the pupils were equal and reacted normally; that there were no pareses, but that the tongue showed a constant tremor. It was also noted that there was hyperresonance over the whole left side of the chest; that the area of cardiac dulness was absent; that the apex beat could be felt to the right of the sternum; that the other organs were normal in position. Three days after admission he began to complain of abdominal pain; he developed fever, and had an enlarged spleen, and three days later rose-colored spots appeared upon the abdomen. He was therefore transferred to the medical ward. Upon admission the following notes were made:

"The left side appears to be larger than the right; no cardiac impulse is visible on palpation to the left of the sternum, but there is distinct impulse visible in the third, fourth, and fifth interspaces to the right of the sternum. The percussion note over the left side is tympanitic below and resonant above the second interspace. The tympanitic note extends from the midsternal line to the midaxillary line. On the right side there is a little fulness in the upper portion of the chest, and tenderness as far down as the second rib.

The percussion note is resonant, but not tympanitic. In the third, fourth, and fifth interspaces there is an area of dulness which is continuous with the liver dulness, extending from the midsternal to the parasternal line. The respiratory sounds are absent on the left side below the level of the fourth rib anteriorly, and below the level of the third rib laterally. Below this point a considerable amount of gurgling and bubbling can usually be heard. Above the fourth rib the respiratory sounds are vesicular, but harsh. The heart sounds are faint on the left side and loud on the right side, and the second sound is distinctly accentuated. The patient shows general cyanosis."

History in the Medical Ward. One week before admission, after exposure, the patient had a chill, accompanied by vomiting. Since then he had been feverish and chilly, and had had several attacks of epistaxis, and cough with blood-streaked expectoration. There had been some pain in the abdomen.

April 10, 1902, the following notes were made: "The tongue is moist and tremulous; the pulse rapid, weak, and regular. There is bulging in the intercostal spaces on the left side, and a visible cardiac impulse in the third, fourth, and fifth interspaces just to the right of the sternum. Percussion resonance is normal on the left side as far as the second rib; below this there is distinct tympany extending from the midsternal to the midaxillary line. If the patient inspires deeply the upper border of the tympanitic area descends about an inch. There is a slight prominence in the right infraclavicular space which is tender. The area of cardiac dulness extends one inch to the right of the sternum, and from the lower border of the third rib to the hepatic dulness. The percussion note over the rest of the right side is clear. The breath sounds are very faint on the left side below the third rib. Over the tympanitic area bubbling, gurgling, and splashing are heard. When the patient swallows water and the stethoscope is placed over the tympanitic area the gurgling sounds are clearly transmitted to the ear. The heart sounds are faint on the left side, but loud on the right, and there is accentuation of the second sound. In the lower part of the left side anteriorly there is a to-and-fro sound resembling friction rub. When the patient is placed upon the right side the area of tympany extends as far back as the posterior axillary line. There is dulness along the left border of the sternum. Posteriorly the breath sounds can be heard indistinctly at the base of the left chest. The coin test is not obtained."

The following extracts from the notes relate most intimately to the physical condition:

April 11*th.* Two exploratory punctures were made, but were entirely negative. It felt as though the needle were in a hollow cavity. The course of the disease has been steadily progressive. From time to time the patient has had severe hemorrhage from

the bowels; the diazo and Widal reactions have been positive, and the eruption is typical. The respirations are never very rapid.

14*th*. There seems to be a slight but appreciable movement of the area of dulness to the right of the sternum, farther to the right and slightly upward.

15*th*. Auscultatory percussion over the tympanitic area indicates that it is apparently continuous with the abdominal cavity. When the stethoscope is placed over this area, and the abdomen tapped, the sound is clearly transmitted, unless the tapping is made over the area of liver dulness, over the right lung, or above the third rib.

25*th*. The patient has had an epileptic attack. For the preceding two days he has had dyspnœa.

After this date he continued to grow worse, extensive and uncontrollable bed-sores developed, he expectorated large quantities of rusty sputum, the respiration, increased in frequency, and the heart was apparently displaced slightly farther to the right. The area of tympany remained unchanged. Posteriorly there was a slight area of dulness extending from the angle of the scapula to the base of the lung. This area moved slightly when the patient was rolled upon the right side. Over it the breath sounds were harsher than over the tympanitic area. The pulse became weaker, and at 8.45 on the morning of April 30, 1902, the patient died. The blood count was made only once, when there were 4000 leukocytes. Throughout the course of the disease he had suffered from excessive thirst. Litten's phenomenon was never satisfactorily obtained, partly on account of the serious condition of the patient.

Immediately after death Dr. G. E. Pfahler took a skiagram of the thorax and abdomen of the patient. This was not developed until after the autopsy, but would alone have rendered the nature of the condition perfectly clear.

The diagnosis of this condition presented many difficulties, chiefly on account of our unfamiliarity with the condition. At various times we suspected pneumothorax, diaphragmatic hernia, pulmonary cavity, and high position of the diaphragm. In a subsequent study of the literature it appeared that we should also have considered subdiaphragmatic pyopneumothorax and unilateral paralysis of the phrenic nerve. In favor of *pneumothorax* was the movable dulness on the left side posteriorly. This, however, was always doubtful, and the condition appeared to be excluded by the absence of dyspnœa, the equal expansion of the two sides during respiration, the negative coin test, the movement of the upper border of the tympanitic area during respiration, failure of the supposed fluid to increase during the course of the disease, and the absence of leukocytosis. A tympanitic note, moreover, is rather uncommon over a pneumothorax. The diagnosis of *diaphragmatic hernia* was concurred in by Dr. Riesman, who also saw the case.

In favor of this was the area of tympany in the lower portion of the left chest, the dislocation of the heart to the right, the loud gurgling heard in the tympanitic area when the patient swallowed water, and the excessive thirst. Against it was the absence of dyspnœa, the equal expansion of the two sides, the area of dulness posteriorly, and particularly the distinct respiratory movement of the upper border of the tympany. Cavity in the lower portion

The skiagram was taken with the patient's back to the plate. It is, therefore, as if the patient were standing with his back toward the spectator. On the right side is seen the heart, and below that the convex upper surface of the liver, indicating the position of the right half of the diaphragm. On the left side there is, next to the spinal column, a narrow band of shadow indicating the left border of the heart. Then, arising from the vertebral end of the seventh rib, arching to the left and upward as far as the fifth rib and axillary line there is a convex shadow indicating the position of the left half of the diaphragm. The large dark mass just below this was found at the autopsy to be a huge coagulum of milk lying in the stomach. Below this the attachment of the diaphragm can be seen, represented by a broader line convex on the upper surface, and below this and to the left of the vertebral column a lighter shadow, indicating the position of the spleen. (The skiagram was taken by Dr. G. E. Pfahler.)

of the left lung was also suggested, but there was neither bronchophony nor pectoriloquy over the tympanitic area, no paroxysms of profuse expectoration, and in cavity formation dislocation of the heart, excepting toward the cavity, is exceedingly uncommon.

High position of the diaphragm accounted satisfactorily for the dislocation of the heart, the respiratory movement of the upper border of the tympany, and for the gurgling heard over the tympanitic area, particularly if the water was swallowed during auscultation. We did not understand why there should have been a supposed movable dulness posteriorly, nor could we account for the friction sound occasionally heard in the fifth interspace near the left midaxillary line. Nor was there any obvious cause why the diaphragm and with it the abdominal viscera should have been displaced. It was therefore supposed that it might be congenital, possibly one of the forms of physical anomaly sometimes associated with epilepsy. Hypoplasia of the left lung was not suspected during the patient's life.

The autopsy was made by Dr. Prince five hours after the patient had died. The pathological diagnosis was: Congenital malformation and hypoplasia of the left lung, compensatory distention of the left half of the diaphragm, splenic tumor with septic infarction, enlargement of the liver, acute nephritis, and enteric fever. The most important parts of the notes are as follows:

There was a large bed-sore over the sacrum and one over the seventh cervical vertebra; also beginning bed-sores on each heel. Abdominal cavity: The lower edge of the stomach could be seen just beneath the costal border; its position was almost vertical. The spleen was greatly enlarged; its lower border was at the level of the border of the ribs anteriorly. The left kidney was in its normal position. The tail of the pancreas pointed vertically upward along the left side of the sternum. The diaphragm turned sharply upward just behind the xiphoid cartilage, and on the left side reached to the third intercostal space. On the right side it reached to the fifth rib. Thoracic cavity: The left pleural cavity was abnormally small, the dome of the diaphragm reaching high into the chest. The heart was displaced toward the right and extended 5 cm. to the right of the midsternal line, and from the third to the sixth ribs. The left lung was abnormally small; the length of the outer edge was 16 cm. and of the anterior edge 13 cm. Its greatest width was 18 cm. The lower lobe was imperfectly divided into three small lobes; there were some areas of atelectasis posteriorly and slight emphysema anteriorly. The right lung was apparently normal in size. The middle lobe was imperfectly separated from the upper. It was slightly emphysematous and showed a few small areas of atelectasis. The spleen measured 15 x 6 cm. The substance was firm; there was a large, softened, anæmic infarct on the greater convexity, and a second somewhat smaller infarct a short distance from this. It was placed close to the spinal column, with its long axis vertical and extending upward to the level of the sixth dorsal vertebra. Microscopically the diaphragm contained muscular fibres.

The physical signs were satisfactorily explained by the autopsy. The left lung was less than half its normal size, and the small areas of atelectasis at the surface did not permit, in the presence of emphysema of the other portion, of the supposition that it had ever been compressed. It must, therefore, have been hypoplastic, and coincidently with this hypoplasia the diaphragm had risen to fill the left pleural cavity. The diaphragm was moderately thin, but microscopically was found to contain a fair amount of muscular tissue, which accounted satisfactorily for the respiratory movement. The area of dulness to the left of the sternum posteriorly was evidently due to the greatly enlarged spleen which was found in this situation, and it is entirely possible that in rolling the patient from side to side this large and heavy organ may have shifted enough in position to give the impression of movable dulness. It is also possible that the rubbing of the spleen against the wall of the abdomen gave rise to the friction sound. No other cause was found at the autopsy.

The condition was unquestionably congenital, for, in addition to the peculiar deformity of the left lung, it seems likely that the dislocation of the tail of the pancreas upward must have occurred during embryonal existence.

The following list of cases of this condition is as complete as we could make it. Unfortunately it was impossible to verify all of the references, and some of the cases are so incompletely reported in abstract that their nature is doubtful:

Case I.—Jean Louis Petit mentions the case of a woman who had had attacks of asthma for some years, which were usually relieved by eating. She died of inflammation of the stomach (peritonitis?), and at the autopsy a tumor the size of a small gourd was found projecting into the left pleural cavity. It contained the stomach, colon, and great omentum. Its wall was composed of the pleura, the peritoneum, and the diaphragm. He describes it as a form of diaphragmatic hernia.

Case II.—Lawrence reports the results of an autopsy in which the liver was found displaced in the right lumbar region; the diaphragm on the left side reached to the second rib, forming a sac which contained a greatly dilated stomach. The left lung was reduced to the size of a fist, and the left half of the diaphragm formed a thin, flaccid septum which was continuous with the muscle on the opposite side. The right half was normal. The position of the heart is not mentioned.

Laacher, in his article, mentions the two following cases:

Case III.—Pyl describes the findings of an autopsy upon a newborn infant. On the left side the diaphragm was thin and dilated as if it were a hernial sac. This sac contained the stomach, spleen, ileum, and the curve of the colon. The heart was pushed to the right. He regards it as a hernia vera.

CASE IV.—Meckel describes a female fœtus, the left side of whose diaphragm was dilated into a sac containing the stomach, the jejunoileum, the cæcum, the colon, and the spleen.

CASE V.—Froriep mentions the case of a girl who died at the age of nineteen years. She often had dyspnœa and vomiting and other digestive disturbances. From the fourteenth year she had had obstinate constipation. At the autopsy the left half of the diaphragm appeared as a sac reaching upward into the pleural cavity, and containing the stomach. The heart was pushed considerably to the right. The diaphragm was abnormally thin, and in the neighborhood of the mediastinum was transformed into transparent cellular tissue. This abnormal thinness appeared to be congenital, for no other cause, such as inflammation of the diaphragm or of the diaphragmatic pleura, existed.

CASE VI.—Marsh reports the case of a man who had a severe shock followed by a sense of something giving way, and then cough, dyspnœa, and pain in the left side of the chest. At the autopsy the left half of the diaphragm arched into the left pleural cavity as high as the level of the left intercostal space. The stomach was concealed beneath the ribs. The liver lay below and in front of it. The right half of the diaphragm was normal. The left half contained a few muscular fibres; the left crus was about half the size of the other. The lungs showed the changes of acute croupous pneumonia, but were otherwise normal; their size is not mentioned.

CASE VII.—Thoma describes the findings at the autopsy of a man of seventy-five years who had died of peritonitis. The diaphragm reached to the fifth rib on the right side and to the third interspace on the left side. The left half formed a large sac containing the stomach, spleen, the left lobe of the liver, and part of the colon, all of which were displaced upward. The apex of the heart was in the median line just above the xiphoid cartilage. The upper lobe of the left lung was large and extended far to the right; the lower lobe was very small, so that the whole lung was shorter. The right lung was deeply grooved on the anterior surface to receive the heart. The spleen was moderately and the stomach greatly enlarged. Thoma naturally regards the condition as congenital, particularly as other forms of congenital hernia were present.

CASE VIII.—Crispino reports, as an instance of congenital dextrocardia, the case of a woman fifty-five years of age. There was no deformity of the chest. There was a small left inguinal hernia. The apex of the heart could be felt in the fifth interspace in the right anterior axillary line; the heart dulness commenced at a point above the fourth rib and extended from the right border of the sternum to a line between the right nipple line and the right anterior axillary line. The lungs were entirely negative. Auscultation of the heart revealed only slight arrhythmia. He expressly

states that the spleen, stomach, liver, etc., were in their normal positions. But he then describes a radiograph which showed that the heart was greatly displaced to the right, and that the left half of the diaphragm was higher than the right, the difference being about four inches. It moved easily and synchronously with the heart. The left lung appeared to be short, but very broad.

CASE IX.—Kronig reported very briefly before the Berlin Medical Society, as an instance of dextrocardia, the case of a man, forty-eight years of age, who died of pneumonia, and in whom, before death, a diagnosis of dextrocardia had been made. At the autopsy the heart was found dislocated to the right, but not otherwise anomalous. The left lung was very broad, and the relation of the lobes was altered so that the upper lobe lay directly in front of the lower.

CASE X.—Tennant reports the case of a man of sixty years who had had syphilis and psoriasis, and died as the result of double pneumonia. There was nothing in his clinical history to indicate disease of the gastrointestinal tract, and apparently nothing unusual had been discovered in the physical signs. The condition was found during dissection. There was no obvious external deformity excepting large bilateral inguinal hernias, which were not congenital. On the right the diaphragm extended to the fourth interspace; on the left the anterior third of the diaphragm was normal, then it bent sharply upward, forming an edge from the tenth rib to the central tendon, and a pouch that extended to the second interspace. This contained a distended stomach and coils of the transverse colon, which was fifty-five inches long. The membrane forming the pouch was thin and translucent, and contained only a few muscular fibres examined macroscopically. The apex of the heart was three inches to the right and one and a half inches to the right of the median line; the base was normal. The lungs were also normal and equal in size. The left phrenic nerve did not show any obvious degeneration. Tennant believes that the condition must either have been congenital or acquired as a result of the degeneration of the muscle of the diaphragm or the terminations of the phrenic nerve.

CASE XI.—Doering reports the case of a man of sixty years who had always performed severe manual labor. His thorax was slightly barrel-shaped but symmetrical, and there was possibly a slight diminution of the respiratory excursion on the left side posteriorly. Otherwise it was normal. Litten's phenomenon could not be satisfactorily determined. Percussion of the left side anteriorly showed slight impairment of resonance as far as the third rib; below this point there was a loud tympanitic note. Posteriorly the area of tympany commenced at the level of the fourth spine of the vertebra. On the right side percussion was entirely normal. The cardiac pulsation could be seen and felt in the fourth inter-

space, a finger's breadth to the inner side of the right nipple line. The dulness extended from one finger's breadth to the left of the left border of the sternum as far as the right nipple line, to the level of the third rib and the liver. Auscultation showed vesicular respiration at the apex of the left lung, loud intestinal gurgling over the tympanitic area, normal vesicular respiration over the right lung, and an accentuated pulmonic second. A diagnosis was made of dislocation of the heart and retraction of the left lung. At the autopsy the diaphragm was found reaching to the sixth rib on the right side, then bending sharply upward at the suspensory ligament of the liver, and forming a large sac the apex of which reached to the upper border of the third rib. The heart was entirely dislocated to the right, but was in itself normal. There were no pleural or pericardial adhesions. The lung consisted of three lobes. The left lung was small, measuring 14 cm. vertically, 12 cm. in breadth, and 6 cm. in length. The tissue was normal excepting a slight pigmented induration of parts of the middle and lower lobes. The right bronchus was larger than the left. There were no bronchiectases. The sac formed by the diaphragm contained portions of the stomach and colon and a large mass of intestinal loops. The spleen was small and in its normal position. The microscopic examination of the diaphragm showed that it consisted of three layers, one of the pleura, another of the true diaphragm, and between them a layer of laminated connective tissue. The muscular fibres were greatly diminished, and in the upper third of the sac were entirely absent. The left lung was normal.

Case XII.—In 1901 Widenmann reported as an example of diaphragmatic hernia diagnosed during life the case of a man, forty-eight years of age, who had had four attacks of pleurisy. The last attack had commenced with profuse hemorrhage from the stomach. He was transferred to the hospital, where it was found that the left thorax was slightly prominent above and slightly retracted below. The expansion was almost equal. On the left side from the second rib to the fourth rib there was a hyperresonant note, and from the fourth rib downward tympany. Posteriorly there was loud tympany from the angle of the scapula to the level of the ninth spinous process, and below this dulness which continued to the upper border of the spleen. In the tympanitic area the breath sounds were practically absent, and on this account the intestinal gurgling could clearly be heard. Hippocratic succussion could be obtained after taking food. The heart was dislocated to the right; the area of dulness changed considerably with the position. The stomach contents were normal. When the stomach was distended there was no prominence in the left hypochondriac region; the area of tympany rose one interspace higher in the left chest. In order to confirm the diagnosis the patient was studied by the aid of the Roentgen rays, and a large clear space seen in the

lower border of the left thorax, bounded above by a line convex upwardly. On the first examination this moved with respiration, and a diagnosis of high position of the left diaphragm was made.

Later a transverse line below the clear area could be seen moving with the respiratory excursion, and a diagnosis was made of diaphragmatic hernia, the supposition being that the opening was in the posterior portion of the diaphragm, and that the left lobe of the liver had also passed through it, accounting for the area of dulness posteriorly. Notes taken twenty-six years before in the military hospital revealed practically the same condition. A year later Glaser reported the same case. The physical signs were practically the same. Litten's phenomenon could not be observed. If the stomach were filled with water, dulness appeared in the lower portion of the tympanitic area. The patient again had hæmatemesis, and incarceration of diaphragmatic hernia was diagnosed. An operation was performed, at which high position of the diaphragm was discovered. The spleen was dislocated upward, lying to the left and back of the fundus. The patient recovered from the operation, but a year later died from carcinoma of the tongue. At the autopsy the right apex of the diaphragm was found at the lower border of the fifth rib; the left apex at the level of the third interspace. The left half of the diaphragm contained the stomach and some intestinal loops. When the stomach was inflated the apex of the diaphragm on the left side rose to the level of the second interspace. The colon, duodenum, liver, and kidneys were normal in position; the spleen was dislocated upward; the heart was to the right. The left lung consisted of two lobes, the upper covering the lower. Benda examined the diaphragm and found what was apparently a fatty degeneration of the muscular fibres. Fraenkel, in whose service the case occurred, therefore regards it as an acquired high position of the diaphragm caused by a pseudohypertrophic lipomatosis of the diaphragmatic muscle.

In view of Widenmann's discovery of the record that showed that at the age of twenty-two years practically the same condition existed, the disease must have commenced in early life. Somewhat similar cases, both as regards the physical signs and the results of Roentgen examination, have been reported by Hirsch and Strüppler. Both have been diagnosed as diaphragmatic hernias, but it is entirely possible that they may really have been cases of eventration of the diaphragm.

In addition to these cases there are several other doubtful cases in the literature: 1 by C. J. Clark, in which it is not clear whether the condition was congenital or acquired; 1 by Abercrombie, 1 by Barlow, and 1 by Fitzgerald and Ernst. The

data are insufficient, and therefore it is not possible to include these cases.

Summarizing these 13 cases we find that 1 was a fœtus; 1 a newborn infant. In 8 the age is given and ranged from nineteen to seventy-five years; in 3 it is merely stated that they were adults; 4 were females, 7 were males, and in 2 the sex was not given.

The clinical symptoms were various. In 1 there was asthma relieved by eating; another, a girl of nineteen, had suffered from vomiting and constipation; a third, a man of fifty, had had a number of attacks of hæmatemesis and symptoms of incarceration of a diaphragmatic hernia for which operation was performed. One patient had inguinal hernia, another bilateral inguinal hernia, and 1 suffered from epilepsy. In 2 cases evidences of peritonitis were found at the autopsy. One died of pneumonia, 1 of typhoid fever, and 1 of carcinoma of the tongue.

In 12 of these cases autopsies have been obtained; 1, the case of Crispino, is still alive, but the diagnosis appears to be established by the radiogram.

In 8 of the 12 cases the diagnosis was not suspected. In 1 a diagnosis of dextrocardia had been made during life, and in 3 the diagnosis was made with a reasonable certainty during life, Doering diagnosing high position of the diaphragm with retraction of the left lung, Glaser having established the dignosis at an exploratory operation, and in our case the diagnosis of high position of the diaphragm having been made.

The condition of the lungs is mentioned in 8 cases. In 2 of these it is stated that they were normal, and in 1 particularly that they were of the same size. In 6 cases they were abnormal. Twice three lobes were present, and the lung was small, and in the remaining 4 cases the upper lobe was relatively much larger than the lower lobe, and its position in relation to it was somewhat anomalous.

The only important conclusion to be deduced from these figures is that eventration of the diaphragm does not interfere with the duration of life, nor does it necessarily produce clinical symptoms.

With this scanty material it would not be justifiable to attempt to draw any very definite conclusions were it not for the fact that all the cases exhibit a very remarkable similarity; indeed, with unessential variations, the description of the autopsy in one case might readily be used for all the others.

Regarding the nature of the condition there is some dispute. Cruveilhier intimates, but does not explicitly state, that in the case of Petit, which he studied, it may have been acquired. The evidence for this was in part the coexistence of peritonitis, but subsequent authors—with, I believe, the exception of Glaser, who derived his views from Fraenkel and Benda—have been inclined to regard it invariably as congenital. In favor of this is the fact

that 2 cases have been recorded occurring in newborn children, as well as the existence in several cases of distinct anomalies of the left lung and the presence in 2 cases of other forms of hernia. There is more difference of opinion as to how the lesion actually occurs. Thoma believes that the lung is chiefly at fault. He urges that weakness of the left side of the diaphragm cannot account for it, as in the fœtus the liver protects both sides. There is a greater tendency on the part of the left lung to congenital malformation in the proportion, he finds, of 49 to 8. He suggests that it is not impossible that eventration might occur on the right side, although hitherto no such case has been recorded. The arguments in favor of this view may be summarized as follows: First, the occurrence of congenital anomalies in the lung; second, the presence of muscular fibres in the diaphragm in several cases examined microscopically; third, the absence of anomalies in the great vessels arising from the heart. The mechanism is, of course, obvious. When the lung is too small the diaphragm rises to fill the space in the pleural cavity, dragging the intestinal contents with it.

The theory that the primary lesion is a congenital deficiency of the muscles of one side of the abdomen has been supported chiefly by Doering. In favor of his view he urges the marked deficiency in the muscular tissue of the affected side of the diaphragm, which has been found in all cases examined microscopically, and the symmetrical condition of the thorax. The mechanism is that the congenitally weak diaphragm fails to expand the lung, which remains atelectatic, and therefore the diaphragm assumes the high position, dragging the viscera with it. Against this view is, of course, the existence of anomalies in the lung; the fact that the lungs have never been found to be atelectatic; and, I believe, the invariable presence of dextrocardia. Glaser suggests as an explanation of his own case that there occurred a dystrophy of one-half of the muscle of the diaphragm, causing it to become weakened and to rise in the thorax as a result of the traction of the lung.

Tennant suggests that in his case there may have been a degenerative condition either of the left half of the diaphragm or of the terminals of the phrenic nerve. It does not appear that microscopic examination was made for the purpose of determining this important point. It is not an exaggeration of a normal condition, for Engel has proven that the diaphragm is normally higher on the right side.

There are, I think, certain points in this condition which require more careful consideration. The symmetrical thorax early attracted attention. Neisser believes that it is due merely to the greater compensation that can occur in the intrathoracic viscera in early childhood, and particularly to the absence of adhesions between

the serous membranes. Doering implies that it is due to the tendency of the skeleton to develop symmetrically when there is no hindrance placed upon it. These views are practically identical. The fact that the diaphragm on the left side is thin and contains few muscular fibres is to my mind of considerable importance. If the primary lesion were merely a hypoplasia of the lung we should expect some evidence of hypertrophy of the muscular tissue as a result of the additional effort to which it was subjected. Such evidence is entirely lacking; indeed, in one case it was noted that even the pillars of the diaphragm were diminished in size. The dextrocardia hardly accords with the theory of primary hypoplasia of the lung. Assuming that the lung was too small, the natural effect of the tug of the diaphragm would be to displace the heart farther to the left. This is not merely a theoretical consideration. Cases have actually been reported in which, as a result of hypoplasia of the left lung, the heart was displaced to the left side. Reinhold has reported 3 cases: First, a woman aged twenty-four, with a symmetrical thorax and an apex in the left axillary line; there was tympany over the lower portion of the thorax; the autopsy showed bronchiectasis of the left lung and dislocation of the heart to the left. Second, a man of fifty-six years, with similar physical signs. Third, a man of thirty-two years, with an apex beat between the posterior axillary line and the angle of the scapula, and tympany over the lower portion of the left chest. Neisser has reported 4 cases: First, a man of forty-two years of age, with symmetrical, barrel-shaped thorax and an apex beat in the left midaxillary line at the level of the third rib; the diaphragm was high on the left side. Second, a boy of nine years, son of the preceding case, had almost identical physical signs. In 2 additional cases reported briefly there were similar physical signs, with the exception that in 1 there was deformity of the thorax. Heyse reports 1 case, a woman of forty-six years. There was no deformity of the thorax, but evidence of deficiency of the left lower lobe and the heart was dislocated far to the left. Sieveking reports a case with autopsy. The left lung was malformed and also showed evidence of an acquired lesion. The heart was displaced to the left.

These cases seem sufficient to establish the contention that there does exist a condition of hypoplasia of the left lung, with or without a high position of the diaphragm, and with dislocation of the heart to the left. In eventration it has generally been supposed that the dextrocardia is purely mechanical, due to the sliding of the heart down the hill produced by the ascent of the left half of the diaphragm, but such a dislocation could hardly occur as a result merely of hypoplasia of the lung, nor is there sufficient evidence in favor of Doering's view of simple weakness in the left half of the diaphragm. Therefore, it seems to me that we are compelled

to assume that the two conditions coexist; in other words, that in the small number of cases of eventration of the diaphragm hitherto reported, with the possible exception of that of Glaser, there has been not merely a congenital hypoplasia of the lung, but also a hypoplasia or dystrophy of the left half of the diaphragm, and that the dislocation of the heart is due less to the movement upward of the diaphragm than to the elastic tension of the right lung. We confess that after reaching this conclusion the pathology of the whole condition is still entirely obscure. We do not know what is the nature of the atrophy of the diaphragm; whether it is due to some lesion of the nervous system, or of the muscle. The cause of the origin of congenital anomalies in the lungs is purely a matter of speculation.

Aside from the pathology of the subject there is but one other point that is of interest, and that is the diagnosis. This is certainly of importance, because in one of the recorded cases, as a result of a mistaken diagnosis of incarcerated diaphragmatic hernia, operation was performed, uselessly of course, but fortunately without disastrous results. The signs in the cases that have been studied clinically have been practically the same. In all there was normal percussion resonance in the upper portion of the left lung; tympany below a certain line, which varied from the second to the fourth ribs; tympany posteriorly, usually from the angle of the scapula down; the ordinary signs of displacement of the heart toward the right; the evidence of the presence of abdominal viscera in the lower portion of the left thorax—that is, gurgling and splashing, especially pronounced when the patient swallowed liquids; often an area of dulness in the lower part when the patient took a considerable amount of food, sometimes associated with distinct Hippocratic succussion. The coin test is negative. Frequently there is an area of dulness posteriorly which appears to be satisfactorily accounted for in 2 cases by the dislocation of the spleen. Litten's phenomenon is usually absent. The movement downward of the area of tympany on deep inspiration was present in 2 cases. The examination with the Roentgen ray in all cases showed that the abdominal viscera were in the lower portion of the left thorax, and this, in 1 certain case, was conclusively proven by introducing a stomach tube filled with mercury, which cast a shadow, and which could be seen coiled up in the position usually occupied by the left lung (Grosser's method). Clinical symptoms may be entirely absent. The gastric disturbances that occur may sometimes possibly be due to other causes; constipation is common enough not to ascribe it to the eventration; but in the case of Widenmann no cause for the hæmatemesis was found, excepting the dislocation of the stomach.

In making the differential diagnosis all those conditions must

be excluded that produce tympany in the lower portion of the left chest. Among these probably the most important is pneumothorax. In Strüppler's case puncture was made nineteen times and in ours twice. On no occasion, fortunately, did the needle pierce the stomach wall. In left pneumothorax the respiratory movement is usually absent; the interspaces are dilated; the percussion note is resonant rather than tympanitic; the coin test is positive. In eventration of the diaphragm the respiratory movements are normal; the note in the lower part of the chest is clear and tympanitic, and, in the upper part, is resonant. There is loud gurgling—especially when the patient drinks water—heard over the lower part of the chest. The two conditions have in common dislocation of the heart to the right; Hippocratic succussion, which may disappear, however, in eventration of the diaphragm if the patient has fasted for a long time. If the stomach is inflated in pneumothorax there is distinct bulging in the left hypochondrium. In eventration this is not likely to occur, or is slight, and there is distinct bulging of the lower part of the thorax. Litten's phenomenon is apparently absent in both conditions. One of the most valuable differential signs is the fact that the upper border of tympany moves with respiration in eventration and does not move in pneumothorax. Examination with the Roentgen ray is decisive as regards the differential diagnosis between these two conditions. The presence of a sound filled with some opaque substance in the lower portion of the right thorax indicates that the stomach has risen in that region.

More difficulty would probably be experienced in the case of subdiaphragmatic pyopneumothorax. This is an exceedingly rare condition, and when it does occur is nearly always found upon the right side. The condition is very acute and the patients ordinarily are in a state of collapse, and as the commonest cause is the rupture of a gastric ulcer, a history pointing to the existence of this lesion can ordinarily be obtained. In the small number of published cases (von Leyden, Neusser) the symptoms have been tympany in the lower portion of the thorax, the upper border of which moves with respiration; the physical signs of movable fluid below this area of tympany; dislocation of the liver or of the spleen downward; a positive coin test, and slight, if any, dislocation of the heart. Presumably gurgling over the area of tympany was absent, because it is not mentioned in any of the cases.

The majority of cases of eventration of the diaphragm have been confounded clinically and pathologically with diaphragmatic hernia. According to Strüppler the diagnosis of this condition has been made correctly but nine times during life, not including those cases in which it was made as a result of an exploratory

incision. It is therefore fair to conclude, in view of the fact that more than 500 cases have been recorded, that it is exceedingly difficult. It is interesting to note that 1 of the 9 cases was that of Widenmann, which was subsequently shown to be one of eventration of the diaphragm.

The majority of analyses of the symptoms that have been published are based upon the study of single cases, and therefore are only of suggestive value. Diaphragmatic hernia usually occurs upon the left side, giving rise to an area of tympany in the lower portion of the thorax, causing displacement of the heart to the right and compression of the left lung. There is gurgling over the tympanitic area, and the physical signs, therefore, must be practically identical with those of eventration of the diaphragm, with the possible exception that the upper border of tympany does not move with respiration. Unfortunately in none of the published cases of diaphragmatic hernia, as well as we have been able to ascertain, has attention been paid to this point. In traumatic cases of diaphragmatic hernia the history of severe injury will be of great assistance. Extraordinary variability in the symptoms is common to both conditions, and the case of Glaser shows that signs of incarceration may occur in cases of eventration. Dysphagia paradoxica has not yet been reported in this condition, and is by no means common in diaphragmatic hernia. There was a history of persistent thirst in our case, and this has been noted in several cases of diaphragmatic hernia. Examination with the *x*-ray has been shown to be inconclusive, although apparently respiratory movement of the convex line bounding the clear area is in favor of eventration of the diaphragm.

Acquired high position of the diaphragm may apparently occur. Glaser inflated two dilated stomachs ad maximum and found that the diaphragm moved upward a full interspace on the left side. It does not appear that idiopathic dilatation of the stomach produces this displacement. Very little is known of the results of disease of the phrenic nerve. Stockton, however, has reported an extraordinary case which he records as phrenic paralysis, in which there was high position of the diaphragm and tympany in the lower portion of the left thorax. It is not stated whether or not the area of tympany moved up and down during respiration or not, but presumably it did not do so. The symptoms that are described strongly suggest eventration of the diaphragm, but the patient made a complete recovery with disappearance of all the physical signs, indicating that the lesion was transient and not congenital. Without a very clear history it would seem practically impossible to differentiate the two conditions. Cavity or dilated bronchus in the left lung could hardly give rise to difficulty in diagnosis. The character of expectoration,

the absence of displacement of the heart, the amphoric respiration, bronchophony, and pectoriloquy would serve almost immediately to make the diagnosis clear.

LITERATURE.

Abercrombie. Edinburgh Medical Transactions. Quoted by Clark.
Bowditch. Boston Medical and Surgical Journal, 1853–54, vol. lx. p. 65.
Barlow. British Med. Jour., January 3, 1880.
Clark, C. J. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1850, p. 106.
Crispino. Riforma Medica, 1900, Nos. 187 to 189.
Cruveilhier. Traité de l'anat. path. générale, Paris, 1849, vol. i. p. 617.
Doering. Deutsche Archiv f. klin. Med., 1902, vol. lxxii. p. 407.
Engel. Wiener klin. Woch., 1868, p. 121.
Froriep. Kleinert's Repertorium, 1837.
Fraenkel. Centralbl. f. innere Med., 1892, p. 1201; 1893, p. 27.
Fitzgerald and Everett. British Med. Jour., September 18, 1900.
Grosser. Wiener klin. Woch., 1899, p. 655.
Glaser. Deutsche Archiv f. klin. Med., 1903, vol. lxxviii. p. 370.
Hirsch. Münchener med. Woch., 1900, p. 996.
Heyse. Deut. med. Woch. 1903, No. 44.
Kronig. Centralbl. f. innere Med., 1898, vol. xix. p. 178.
Kaufmann. Deut. med. Woch., 1887, No. 28.
Laacher. Deutsche Archiv f. klin. Med., 1880, vol. xxvii. p. 268.
Leichtenstein. Ziemssen's Handbuch der Speciellen Path. und Therap., 1876, vol. vii., part ii. p. 439.
Lawrence. Lancet, 1852, vol. ii. p. 327.
Marsh. Lancet, 1867, vol. i. p. 298.
Meckel. Quoted by Laacher.
Neisser. Zeitschrift f. klin. Med., vol. xlii. p. 88.
Neusser. Wiener med. Woch., 1884, p. 1306.
Petit. Traité de maladies chirurg., 1790, vol. ii. p. 234.
Pyl. Quoted by Laacher.
Reinhold. Münchener med. Woch., 1893, p. 845.
Strüppler. Deutsche Archiv f. klin. Med., 1901, vol. vii. p. 1.
Stockton. Buffalo Medical Journal, 1898–99, p. 97.
Sieveking. Münchener med. Woch., 1895, p. 68.
Tennant. Edinburgh Medical Jour., July, 1894, p. 29.
Thoma. Virchow's Archiv, 1882, vol. lxxxviii. p. 515.
Widenmann. Berliner klin. Woch., 1901, No. 11.

REVIEWS.

THE NERVOUS AFFECTIONS OF THE HEART. Being the Morison Lectures delivered before the Royal College of Physicians of Edinburgh in 1902 and 1903. By GEORGE ALEXANDER GIBSON, M.D., D.Sc., F.R.C.P. EDIN., F.R.S.E. Edinburgh and London: Young J. Pentland, 1904.

THE author divides his subject into the two main divisions of sensory and motor disturbances of the heart and devotes to each three lectures. The former division embraces the clinical, pathological, and therapeutic aspects of angina pectoris, and the latter lectures on rate, rhythm, and force. Each lecture is preceded by a brief historical review of the literature and the theories formerly held regarding each subject. A series of diagrams illustrate the text, those which show the distribution of pain to the surface of the body and which indicate the areas of cutaneous hyperæsthesia being especially interesting. A number of sphygmographic tracings illustrate the last three lectures on rate, rhythm, and force, and several of these are particularly instructive, in that they demonstrate the differences which may exist between the radial pulse and the apex beat in a given case.

Professor Gibson departs from the commonly accepted division of angina pectoris into true and false (or pseudo) angina, and argues since angina pectoris is an aggregation of symptoms occurring in various conditions and is not a disease, that it is not logical to make this distinction. He advocates a division into organic and inorganic, and still leaves this point in an unsatisfactory state by saying that "it is not pretended that in the latter class there are no structural changes." It would appear that it would have been better had he chosen two terms which expressed his conception more exactly. Following this classification, he includes neurasthenia, hysteria, reflex agencies, and vasomotor instability among the conditions in which angina pectoris may occur, and adds them with the toxic agencies to the inorganic causes of angina pectoris. His explanation is that in neurasthenia and hysteria the anginous attacks are brought about by debility of the heart and instability of the nervous mechanism. The lectures are the result of such wide reading and experience, and go so fully into the consideration of the physiological and pathological features of cardiac disturbances that the book should be of value to all who are interested in cardiac disease. F. W. S.

THE DISEASES OF SOCIETY. (THE VICE AND CRIME PROBLEM.) By G. FRANK LYDSTON, M.D. Philadelphia and London: J. B. Lippincott Company, 1904.

DR. LYDSTON has written a very interesting and noteworthy book which deserves careful perusal by all thinking Americans. The study of the criminal classes from a purely scientific standpoint has not made the progress in this country which it has in Europe, and this book may be considered as an attempt at a popular presentation of the results of the investigations of scientific criminologists adapted to the circumstances which prevail in this country. It deals with topics which are of vital importance to every citizen, thus the chapters on Lynching, Anarchy, and the Social Evil which not only go into the causes and the reasons why such things exist, but also give what the author aptly calls their therapeutics, throw light on subjects about which while there is much talk there is really very little knowledge. The sections on what the author terms "anarchy" of government, of law, of politics, and of capital and labor are very slashing and radical, and might be criticised as containing too strong enunciations of the author's personal views.

That part of the work which treats of genius and degeneracy is most interesting. The author accepts the view that "genius is abnormal and both the product and the cause of degeneracy," and then proceeds to adduce facts bearing upon these points. He thinks that while genius may not be hereditary the neuropathic constitution which underlies genius is very distinctly an hereditary trait. Of course, in a work of this nature it is impossible to give complete references, or to always supply facts to support statements, but it is to be regretted that the author yields to the prevalent tendency of writers on such subjects to make isolated statements concerning persons, and to give lists of names of great men accompanied by a sweeping statement that they all were this, that, or the other. As an instance, on page 461 the author states: "Cuvier, Victor Hugo, Chopin, Bruno, Comte, Madame de Staël, Swift, Johnson, Cowper, Southey, Shelley, Byron, Carlyle, Goldsmith, Lamb, Poe, Keats, Coleridge, De Quincey, Chatterton, George Eliot, George Sand, Alfred De Musset, Newton, Chateaubriand, Balzac, Chatham, Burns, Dickens—all these beacon-lights of the history of genius showed indubitable evidences of degeneracy." Such allegations are not only misleading but unworthy of a work which attempts the presentation of scientific facts even if in a popular manner. Dr. Lydston has occupied official positions which have brought him into intimate contact with criminals of all classes and descriptions, and his observations upon these unfortunates are, therefore, entitled to respect. He disapproves most thoroughly of the prison system which prevails in most parts of the United States and of the method of administering justice. He is opposed to

capital punishment, and strongly in favor of the indeterminate sentence. Dr. Lydston is a very thorough-going materialist and also a man of strong convictions who has no hesitation in expressing them. Accordingly, as might be expected, he runs counter to many of the cherished beliefs and opinions of society. The style in which he expresses himself on such topics is at times somewhat florid, and occasionally almost offensive; but we can readily pardon the author when we consider what a really useful book he has produced. It should be read by every thoughtful citizen, and we are sure that even though he may differ from some of the author's conclusions, he will nevertheless find much food for thought in the facts he presents. F. R. P.

THE SURGICAL TREATMENT OF BRIGHT'S DISEASE. By GEORGE M. EDEBOHLS, A.M., M.D., LL.D., Professor of the Diseases of Women in the New York Post-graduate Medical School and Hospital; Consulting Surgeon to St. Francis' Hospital, New York; Consulting Gynecologist to St. John's Riverside Hospital, Yonkers, N. Y., and to the Nyack Hospital, Nyack, N. Y., etc. New York: Frank F. Lisiecki, 1904.

PROBABLY none of the newer surgical fields has excited more general interest in the surgical and medical worlds than the treatment of Bright's disease by operation, and, therefore, this volume is one which is sure to be eagerly read. The most prolific writer on this subject has been Edebohls. The present book is divided into two portions, the first consisting of 142 pages, being the various papers contributed by Edebohls; the latter portion, consisting of 178 pages, is comprised of a minute report of 72 cases which the author had operated upon up to the end of the year 1903, and his conclusions regarding the results. The work covers the whole history of the operative treatment for acute and chronic Bright's disease, and is most interesting and instructive.

Edebohls is naturally more enthusiastic than other surgeons who have had less experience and a higher mortality, but, nevertheless, he shows great fairness, and does not make any rash claims for the operation of decapsulation. He says that the operation is still on trial and must be judged by the ultimate results; that sufficient time has not elapsed since the introduction of the procedure to enable us to give a final judgment. A review of the cases reported must impress the reader with the undoubted position this operation should hold in surgery. It has unquestionably saved lives when nothing else would, and probably it only remains for us to learn how complete and lasting the cure is and to select the proper cases for its employment. Of the 72 cases detailed, but 7 patients died as a

result of the operation. This mortality is strikingly low, when one considers that 16 of the cases reported were patients who were operated upon when death was imminent, due to the late stage of the disease. Seventeen of the cases are reported as cured, 20 as greatly improved, and none any the worse for the operation.

This is a book which we can heartily recommend, not only to every surgeon, but to every internist. J. H. G.

A Treatise on Bright's Disease and Diabetes, with Especial Reference to Pathology and Therapeutics. By James Tyson, M.D., Professor of Medicine in the University of Pennsylvania; one of the Physicians to the Pennsylvania Hospital; Fellow of the College of Physicians of Philadelphia; Member of the Association of American Physicians, etc. Second edition. Illustrated. Including a section on the Ocular Changes in Bright's Disease and in Diabetes by George E. de Schweinitz, M.D., Professor of Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Philadelphia Hospital; Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases, etc. Philadelphia: P. Blakiston's Son & Co., 1904.

As stated in the preface, this is a new edition and not a new book, and while much of the matter from the former edition of 1881 has been retained, considerable new material has been added. Entire new sections on the optical changes in Bright's disease and in diabetes mellitus by Professor de Schweinitz, and sections on acute interstitial nephritis and the dietetic treatment of nephritis are to be found. An introductory consideration of the histology and physiology of the kidney has been retained from the former edition, and very wisely, for this makes more clear the statement of the morbid conditions in the discussions of these in relation to the various forms of Bright's disease.

The illustrations and plates are of excellent workmanship, being far superior to most productions of this nature, and the work in the text is equally commendable. To each form of nephritis is devoted a separate section, and the features of each are treated at considerable length.

The section on the diet in nephritis is of especial interest, since there has been some little confusion and difference of opinion in regard to the measures advocated by various authorities. Professor Tyson's views differ from some of the accepted opinions in two particulars mainly. In referring to permitting meat in the diet in nephritis, he states that many are of the opinion that white meat (poultry, game, etc.), is essentially different from the red or dark

meats (beef, mutton, etc.), and that patients are often permitted to eat large quantities of the former, greatly to the detriment of their health. He says that there is not a great deal of difference in the amount of proteids in the various kinds of meats, and that any meat may be injurious, except in small quantities and in cases where the kidneys' function has been fairly well re-established. He also points out the fallacy of using large draughts of water, mineral or plain, in nephritis. It is not generally understood that while the total quantity of urine may be increased by this "flushing" of the kidneys, the percentage of urea is relatively lowered, and the actual amount of urea excreted is not materially increased.

The section on acute interstitial nephritis is devoted to the consideration of a distinct form of Bright's disease, which, while not recently discovered, has been considerably studied in late years, particularly by Councilman. Its pathological distinction, briefly stated, consists in a collection of leukocytes in the connective tissue of the kidney independent of change in the epithelium, and these masses of leukocytes do not tend to become purulent. The condition occurs in the acute infectious diseases, is generally fatal, and its symptoms differ from those of the other varieties of nephritis only in its sudden onset and the very early occurrence of œdema. This form of nephritis is either very briefly alluded to or entirely omitted from articles on nephritis in most of the text-books.

A careful description of the gross and minute pathological changes in the kidney in each form of Bright's disease and the full account of the symptomatology, etiology, complications, and treatment make the book one of especial value in the study of a disease so frequently met and so difficult to treat. Its value is increased when Dr. Tyson's wide experience in the study and treatment of Bright's disease is taken into consideration.

Articles on diabetes mellitus and insipidus are also to be found in the volume, and are included because of the fact that one who makes a specialty of diseases of the kidney sees also many cases of diabetes, and in this way Dr. Tyson has been unusually fortunate in opportunities to study these conditions. These subjects are as skilfully handled and are of as much practical value as are the other subjects treated in the book. The section on diabetes mellitus contains a table, compiled by Dr. Daniel W. Fetterolf, showing the comparative analyses of a number of the products especially prepared for diabetics and much lauded and advertised on account of the small quantities of carbohydrates they are supposed to contain. Other tables showing the increased frequency of diabetes, as indicated by the death rate for this and other diseases, are also of interest. The remarks about diabetic coma, its causation and treatment, are valuable, and the discussion of the relative merits of the different mineral waters used in diabetes should prove useful.

It is to be regretted that the author did not include sections on

tuberculosis of the kidney and nephrolithiasis, and also that in describing the treatment of chronic nephritis he makes no mention of the operation of decapsulation of the kidney as advocated and practised by Edebohls. F. W. S.

THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS. Vol. VIII. MATERIA MEDICA AND THERAPEUTICS, PREVENTIVE MEDICINE. CLIMATOLOGY, SUBJECTIVE THERAPEUTICS, FORENSIC MEDICINE. By GUSTAVUS P. HEAD, M.D., Professor of Laryngology and Rhinology, Chicago Post-graduate Medical School. 344 pages. Chicago: The Year Book Publishers, 1904.

THIS volume, edited by Satler, Favill, Bridge, Brower, and Moyer, presents a rather brief but discriminating review of the progress in these departments during the past year. Its merit lies in the convenience of size and division of subjects, and it should be useful for the general practitioner, while the specialist will find the important advances chronicled. We note marked improvement over its predecessors. R. W. W.

OUTLINES OF PHYSIOLOGICAL CHEMISTRY. By S. P. BEEBE, PH.D., Physiological Chemist to the Huntington Fund for Cancer Research, and B. H. BUXTON, M.D., Professor of Experimental Pathology, Cornell Medical College. New York: The MacMillan Company. London: MacMillan & Co., Ltd., 1904.

THIS book of 195 pages sets forth the principal chemical compounds found in the human body, and shows their relationship to other organic substances. Some knowledge of inorganic chemistry is presupposed, but in order to carry the reader on from fundamental facts, the first chapter deals with the theory of solutions and ionization. The subject of organic chemistry is then briefly reviewed, covering the first hundred pages of the book. Here the relations of the organic substances to physiological processes is seldom mentioned, and this part of the work differs very little from what one expects to find in a brief work on organic chemistry.

The last three chapters are on the proteids, enzymes, and disease and immunity. Here chemical processes of physiology are brought out briefly. The work gives the impression of being somewhat overcondensed. The style, however, is clear, concise, and to the point. Many diagrams help to make clear the chemical formulæ in which the book abounds.

As a text-book it will no doubt be of use, since it briefly covers ground important in medical education. W. T. L.

HEALTH, STRENGTH, AND POWER. By DUDLEY ALLEN SARGENT, A.M., Sc.D., M.D., Director of Hemenway Gymnasium, Harvard University. Illustrated. New York and Boston: H. M. Caldwell Co.

THE author gives an impressive argument in favor of regular systematic exercise by people of the industrial class, who disregard or are in ignorance of the vital importance of the development of all the muscles. He cites the deterioration of the physique of the British recruits at the time of the South African War, and very reasonably states that this physical decadence should alarm all civilized nations. To check this, which is directly traceable to modern conditions in the cities, he advocates the establishment of gymnasia which will attract those persons who are put to work at an early age and who, from the increasing specialization of their work, are accustomed to use but a few groups of muscles. It is evident that many orthopedic conditions could be prevented or cured if exercises were undertaken which would give a symmetrical development and correct faulty postures.

Full advice is given in regard to bathing, clothing, diet, and rest; and since on all these points erroneous opinions are commonly held, the instructions given should be of great value. Separate chapters are devoted to the discussion of exercise for children, women, the middle-aged, and the old, and also to methods for the treatment of obesity and leanness.

Fifty-six forms of exercise are described, and Dr. Sargent has adopted the plan of giving each a character and name which connects it with some occupation or sport, as wood-chopping, throwing a ball. To this adds the elements of a game, and is undoubtedly [illegible] for children, it seems to make the exercises more cumbersome and complicated than is required for the muscular movements aimed at, and thus makes them less suitable for adults.

An excellent feature of the system is the recognition of the necessity for relaxation of the muscles and proper attention to respiration, which is in marked contrast to some of the recently advocated systems.

F. W. S.

REGIONAL MINOR SURGERY: Describing the Treatment of those Conditions Daily Encountered by the General Practitioner. By GEORGE GRAY VAN SCHAICK, M.D., Consulting Surgeon to the French Hospital, New York. Second edition. New York: International Journal of Surgery Co.

WE are glad to see the second edition of this little book, which fills its purpose in a very satisfactory manner. In a review of the previous edition, we had an opportunity to mention its good points, so

that the repetition of them is unnecessary. We can, however, repeat that the general practitioner doing occasional surgery, and even the experienced surgeon, will find in it many valuable suggestions in the treatment of minor surgical conditions. J. H. G.

MEDICAL LABORATORY METHODS AND TESTS. By HERBERT FRENCH, M.A., M.D. (Oxon.), M.R.C.P. (Lond.), Medical Registrar, Guy's Hospital; Gillson Scholar, Society of Apothecaries of London; Radcliffe Travelling Fellow, Oxford University. Chicago: W. T. Keener & Co., 1904.

THIS small book of 150 pages contains briefly the principal simple laboratory tests that are made upon urine, blood, sputum, pus, gastric contents, and feces, together with short accounts of the microscopic examination in skin diseases, examinations of serous exudates, cerebrospinal and cystic fluids, and tests for the commoner poisons. In order to include so much in so small a book, a tabulated method is employed. The various methods are briefly but clearly given in a well-arranged manner. The relative importance and sources of error in these methods are mentioned, but explanations of the tests are largely omitted. The book should serve as a handy reminder to the laboratory worker of how the various common laboratory tests are made.

The book contains a number of illustrations taken apparently from free-hand drawings, and which often convey very little meaning. Otherwise, the book is all it claims to be. The statements contained in it are, for the most part, correct, but the few errors that we have found are largely faults of omission rather than commission.

G. C. R.

A PRACTICAL TREATISE ON GENITOURINARY AND VENEREAL DISEASES AND SYPHILIS. By R. W. TAYLOR, A.M., M.D. Third edition, thoroughly revised. New York and Philadelphia: Lea Brothers & Co.

ANY work on venereal diseases from the pen of Dr. Taylor deserves and is bound to receive prompt and eager attention by reason of the long and extensive experience of the author. The present treatise fully justifies one's expectations and ably realizes the wish of the writer to present "a practical, up-to-date, compact treatise." The chapters on Gonorrhœa are thoroughly in accord with the trend of modern thought, and the subject is discussed in a most comprehensive manner, nitrate of silver being considered as the ideal agent for the treatment of the affection. We cannot, how-

ever, agree with the writer's caustic criticism of what he is pleased to term "so-called ideal substitutes for nitrate of silver," some of which are mentioned as "therapeutic curiosities," and all of them as "hybrid" preparations.

As might be expected from the author's great reputation as a syphilographer, the portion of the work devoted to the discussion of syphilis deserves the highest praise. Indeed, it would be hard to find anywhere a more comprehensive treatise on syphilis than the writer has given us here. In the section devoted to genitourinary affections the author's desire to present the subject in a compact form has resulted in some places in a want of completeness in the discussion. As an example of this, may be mentioned the consideration of the operative treatment of hypertrophy of the prostate, to which only three pages of the work are devoted.

The volume as a whole stands in the front rank of treatises on this subject, and should be in the possession of every practitioner of medicine. H. M. C.

DIET IN HEALTH AND DISEASE. By JULIUS FRIEDENWALD, M.D., Clinical Professor of Diseases of the Stomach in the College of Physicians and Surgeons, Baltimore, and JOHN RUHRAH, M.D., Clinical Professor of Diseases of Children in the College of Physicians and Surgeons, Baltimore. Philadelphia, New York, and London: W. B. Saunders & Co., 1905.

THIS volume on dietetics opens with a consideration of the principles of digestion, its chemistry and physiology. The transformation of the different forms of proteids, fats, and carbohydrates in the various parts of the alimentary tract is briefly described. Diet for healthy individuals is then discussed, and such factors as age, climate, and idiosyncrasy are mentioned as influencing the selection of our foods. Foodstuffs are themselves classified in the succeeding chapter and several pages are devoted to the alcoholic beverages. Analyses of most of these are given, with accompanying tables, showing their relative percentages of alcohol and their various chemical constituents.

The sections on milk modification and infant-feeding are noteworthy. In the former, not only is there a good introductory explanation of the general principles of the modification of cows' milk, but also the best-known methods are explained, and a series of formulæ are worked out, the results being tabulated for instant reference.

The latter half of the book takes up the subject of diet in disease. The dietary treatment of the fevers, the infectious diseases, acute and chronic digestive ailments, gout, diabetes, and so on, is repre-

sented systematically. Full information is given as to the hours for feeding, preparation, and administration of the various foods, with lists of a number of useful receipts.

At the end of the volume are figures showing the different cuts of beef, mutton, and pork, and a long list of the chemical composition of American food materials. The book is practical, and the recipes and diet-lists should prove exceedingly useful. A. N.

THE MEDICAL EPITOME SERIES. TOXICOLOGY. A MANUAL FOR STUDENTS AND PRACTITIONERS. By EDWIN WILLES DWIGHT, M.D., Instructor in Legal Medicine, Harvard University. Series edited by VICTOR COX PEDERSEN, A.M., M.D., Instructor in Surgery and Anæsthetist and Instructor in Anæsthesia at the New York Polyclinic Medical School and Hospital, etc. Philadelphia and New York: Lea Brothers & Co.

THIS book of 295 pages consists of a well-arranged brief treatise on toxicology. The various common poisons are systematically treated. Each substance is dealt with somewhat as follows: the method for obtaining it from nature, its properties, commercial and medical use; its criminal use, tonic dose, and action are briefly discussed. Then follows a few paragraphs on the symptoms produced by the poison and their treatment, the post-mortem appearances caused by the various substances, and a few simple tests for their recognition. Actual cases from the literature are used to illustrate the effects of the various poisons—an especially good feature—as it adds much to the interest of the book, and impresses the practical bearing of the subject.

The size of the book should make it suitable for a text-book, and at the end of each chapter several questions are added which might be used for quizzing. The author's style is simple, direct, and clear. The printing of the text is noticeably good. G. C. R.

MEDICAL DIAGNOSIS. SPECIAL DIAGNOSIS OF INTERNAL MEDICINE. A HANDBOOK FOR PHYSICIANS AND STUDENTS. By Dr. WILHELM V. LEUBE, Professor of Medicine and Physician-in-chief to the Julius Hospital at Würzburg. Authorized translation from the sixth German edition. Edited with alterations by JULIUS L. SALINGER, M.D., late Assistant Professor of Clinical Medicine in the Jefferson Medical College; Physician to the Philadelphia Hospital. New York and London: D. Appleton & Co., 1904.

LEUBE'S *Diagnosis* is so well and favorably known through its six German editions that it seems a work of supererogation to go

at length into descriptions. The work, which for the first time has been translated into English by Dr. Salinger, will prove of interest and value to physicians. Certain small additions have been inserted by the translator where, for one or another reason, they seemed necessary; thus, in all respects, bringing the subjects up to the level of the times. Leube's *Diagnosis* is among the foremost, if not the foremost, work on the subject, and this able translation brings it within the range of the non-German-reading students and physicians. J. N. H.

THE PATHOLOGY OF THE EYE. By J. HERBERT PARSONS, B.S., D.Sc. (Lond.), F.R.C.S. (Eng.), Assistant Ophthalmic Surgeon, University College Hospital; Curator and Pathologist, Royal London (Moorfields) Ophthalmic Hospital; Lecturer on Physiological Optics, University College, London. Volume I., Histology. Part I. New York: G. P. Putnam's Sons. London: Hodder & Stoughton, 1904.

THIS is the first complete treatise upon the pathology of the eye that has yet appeared in any language. It is pleasant to have such a book, the first of its kind, in English. Better still, the dress is not only English, but the author has not resorted to mere transcriptions from German monographs; it bears the stamp of original research, besides such a comprehensive grasp of the subject as to justify the author in his undertaking. The work will consist of four volumes. The first volume deals with the pathological histology of the anterior portion of the eyeball as far as the lens, including the bacteriology of the conjunctiva. The normal histology of the parts considered is briefly described. This volume is profusely illustrated from photographs of specimens.

This work is not intended for elementary instruction. General knowledge of histology, both normal and pathological, is required on the part of the reader who would benefit from it; nor should such knowledge have been acquired merely from reading; practical work at first hand in a laboratory is essential for the student who would derive the advantages which this treatise is capable of affording.

At the conclusion of each section a reference-list of papers is appended.

The mechanical execution of the book leaves nothing to be desired.

As the author observes, most of the problems which are here dealt with remain to be solved; the work brings together the data so far as known, and the difficulties to be overcome, and will, no doubt, long remain a standard of reference for all those who concern themselves with the scientific basis of ocular pathology. T. B. S.

A PHILOSOPHY OF THERAPEUTICS. The foundation of which rests on the two postulates: First, that it is the human organism that is the active factor in the healing of the sick, and not drugs, and, second, that there are two therapeutic laws. By ELDRIDGE C. PRICE, M.D. Baltimore: Nunn & Co.

THE author of this volume desires to present to the reader the evil of ultra-conservatism on the part of the advocates of the different schools of medicine. There is considerable space devoted to the consideration of the laws enunciated by Galen and Hahnemann. The fact that there is much good to be found in the ideas of both schools is emphasized. It is a plea for less prejudice and more knowledge. He contends that only by this means can the physician attain the ideal of medical science, "the greatest good to the patient." Unfortunately, the author himself, in many parts of his work, assumes the character of a special pleader for his own views. He is of the opinion that we are too prone to accept statistical results without first viewing the conditions which influence the facts on which the statistics are based. The object of the Baltimore Medical Investigation Club is mentioned in this connection, and the investigation of several drugs by it cited as examples of the proper method of procedure. The style is rather wordy, and there are some typographical errors. A. N.

NOTHNAGEL'S ENCYCLOPEDIA OF PRACTICAL MEDICINE. TUBERCULOSIS AND ACUTE GENERAL MILIARY TUBERCULOSIS. By Prof. Dr. G. CORNET, of Berlin. Edited, with additions, by WALTER B. JAMES, M.D., Professor of the Practice of Medicine in the College of Physicians and Surgeons (Columbia University), New York. Authorized translation from the German, under the editorial supervision of ALFRED STENGEL, M.D., Professor of Clinical Medicine in the University of Pennsylvania. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

THIS is the seventh volume of the Nothnagel series and is devoted to the study of tuberculosis, including practically all that is known of the subject from every point of view.

The work is divided into two parts—I. Tuberculosis, and II. Acute General Miliary Tuberculosis—and these two parts are in turn subdivided into many interesting chapters. In selecting for particular mention any chapter to the exclusion of others in this excellent book one is, in truth, making unfair distinctions, but we may perhaps be forgiven in so doing, on the ground that these particular chapters are of great popular medical and lay interest. Such chapters as those on the Tubercle Bacillus, with reference to morphology,

biology, chemistry, etc.; the modes of Invasion, Infection, Predisposition, and Prophylaxis are full of the very latest scientific thought presented in the clearest terms.

Professor Cornet takes a very strong position in regard to the frequency of infection as the agent in the spread of this widely fatal disease. In the course of his argument, in a most original manner, he points out many fallacies in statistics which might lend an undue weight to either the theory of predisposition or that of heredity, but which, when properly interpreted, serve but to strengthen the view that infection plays the leading rôle, independent of any hereditary or other predisposing cause.

The author's explanation of the frequency of tuberculous lung affections in the wake of certain diseases (whooping-cough, measles, influenza) is plausible and interesting. He ascribes such immunity from phthisis as does exist to the action of the cilia of the epithelial cells lining the air-passages in mechanically brushing back partially inhaled bacteria. In those diseases most commonly followed by phthisis there is a very notable involvement of these same epithelial cells, with the possible result of so crippling the cilia that this repellant function is abrogated. He points out the difficulty of claiming for any organ a greater or less degree of susceptibility, judging merely from the frequency with which this particular organ is attacked, unless the observer is in possession of the knowledge of the mode of invasion. Bovine and human tuberculosis are believed to be caused by the same bacillus, though modified in many respects by its sojourn in the animal or human host.

In discussing prophylaxis, the value of public education in regard to the method of the spread of tuberculosis is pointed out. The individual afflicted with the disease has it within his power to render himself innoxious to his family and the community at large by attention to the one detail which decency and unselfishness should dictate, and that is never to spit on the floor or street, but always, when it becomes necessary, to spit into vessels containing a little fluid. No less interesting are the remaining chapters on Symptoms, Course, Diagnosis, Prognosis, and Therapy. Space, however, will not permit of any but this passing mention.

Never before has this great subject been treated so exhaustively, so clearly, and so intelligently, and Professor Cornet and the editors are to be most heartily congratulated on their work.

J. N. H.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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A Case of Suppurative Myositis Caused by Micrococcus Gonorrhœa (Neisser).—HARRIS and HASKELL (*Johns Hopkins Hospital Bulletin*, December, 1904, p. 395) comment on the multiplicity of the complications of gonorrhœa. They have searched the literature for cases of myositis following this disease, and give abstracts of 7 cases in which a myositis due to the gonococcus was believed to exist. In 6 of these a suppurative myositis was believed to exist, but the bacteriological proof was not sufficiently convincing in any single instance to definitely warrant the opinion that the gonococcus was the cause of the inflammation. Their own case concerned a woman, aged thirty-four years, who since the birth of her last child, eleven months before, had suffered from leucorrhœa. She had had it on previous occasions, and this symptom constituted the only evidence of a gonorrhœal infection. Four weeks before she was admitted to Dr. Osler's wards in the Johns Hopkins Hospital a lump the size of a hen's egg developed in the right calf muscles, giving place later to a diffuse boggy swelling. Several months before she came under observation another swelling, also about the size of a hen's egg, appeared over the lumbosacral region. The abscess in the calf muscles was aspirated and a small amount of pus obtained for bacteriological examination. The pus showed a diplococcus which in its staining and cultural characteristics conformed in every way with those of the gonococcus. The abscess was eventually incised and it was found to contain about one and a half pints of pus. It was situated beneath the gastrocnemius and soleus muscles and extended up into the popliteal space. There had been considerable sloughing of these muscles and their tendons. The abscess in the lumbosacral region was incised and was found to contain about three ounces of pus. It extended up into the erector spinæ muscles on either side, which, with their tendons, showed considerable sloughing. The bacteriological examination of the pus obtained from the abscesses at

operation demonstrated conclusively the presence of the gonococcus. A feature of interest in this case was that there was no co-existent arthritis nor was there any history of its having previously been present.

Abnormalities in the Feces in Disease of the Pancreas.—URY and ALEXANDER (*Deutsche med. Wochenschrift*, 1904, xxx. p. 1311), on the basis of a number of cases and a general survey of the literature, point to the importance of careful examination of the feces in suspected disease of the pancreas, while emphasizing the reserve which must be exercised in interpreting the results. The two points of special interest are the determination of the fat and proteid digestion. There are three possibilities in reference to abnormal fat content of the feces in pancreatic disease. There may be an increased amount of fat with diminished fat splitting, an increased amount of fat with normal splitting, or a normal amount of fat with diminished splitting. It is of great importance in these estimations to regulate accurately the amount and character of the ingested food. Fat kept within the bounds of assimilation is normally well utilized (7 per cent. to 10 per cent. loss), and the limit is high, reaching, with individual variations, about 350 grams for butter. The tolerance for fats is much diminished in disease of the pancreas, and the limit of assimilation relatively low, so that by carefully regulating and changing the diet defects in pancreatic secretion might be recognized at an early stage. Conclusions from the amount of fat in the stools can, however, only be drawn under the following conditions: There should be no jaundice, as absence of bile may of itself cause steatorrhea; the fat should not be administered emulsified, and there should be no diarrhœa. If diarrhœa be present, it may be checked with opium. Even under these precautions the results are of value in diagnosis only when considered in relation to other symptoms. Marked steatorrhœa may occur in disease of the small intestine, with diminution of its absorptive power, and in diseases interfering with the flow of fat through the lacteals, as enlarged mesenteric glands and tuberculous peritonitis. On the other hand, one meets with cases of almost complete destruction or atrophy of the pancreas, in which, during life, fat digestion is perfect. What Ury and Alexander consider almost pathognomonic of pancreatic disease is the discharge of large quantities of liquid fat after the solid, formed feces have been passed. Most cases of steatorrhœa are associated with an excessive number of well-preserved muscle fibres in the stool, although either condition may occur without the other. This is not surprising, in view of the results which have established the independence of fat and proteid digestion. Muscle fibres, of course, occur in normal stools, and to recognize an azotorrhœa, not more than one-half pound of tender meat a day should be allowed. The significance of this condition is likewise relative and occurs in various affections disturbing the movements, secretion, and absorption of the small intestines. A third point of some importance is the evacuation of relatively large quantities of solid feces. Nobel thought the absence of the products of putrefaction was an important diagnostic aid, but Ury and Alexander found a normal amount of aromatic oxyacids in their cases. In diabetes, as a rule, both fat and proteid digestion are well preserved. In certain cases, however, with marked steatorrhœa and azotorrhœa, extensive involvement of the pancreas has been correctly diagnosticated. Naunyn, however, cautions against too great reliance on these symptoms.

Hereditary Syphilis in the Second Generation.—BOECK (*Berliner klin. Wochenschrift*, 1904, xli. p. 968) cites four instances in which the evidence appears to him conclusive that the mother, herself the subject of inherited syphilis, transmitted the taint to her offspring. In the first case the grandmother, at the age of eighteen years, had been treated for secondary lues in 1854; the mother, at the age of two months, for hereditary syphilis in 1860; while the child, aged four to five months, was brought to him with definite lesions of inherited disease in 1889. The mother at this time had Hutchinsonian teeth and typical scars about the mouth, but no sign of secondary lesions. No history could be obtained to support the suspicion of a reinfection. She had had no miscarriages or premature births, but in 1883 was delivered at the lying-in hospital of a healthy child, which she had by another man. Examination at that time revealed no evidence of syphilis either in mother or child. By her husband, the father of the syphilitic infant, she had had, three years before, a perfectly healthy child, and on most careful examination nothing could be found in the father to justify any suspicion of disease. In the second instance the grandmother, when nineteen years of age, was treated at the clinic for a chancre of the lip and subsequently for secondary symptoms. Three years later she gave birth to a dead child. After this, however, she had three children, all of whom lived. The eldest of these presented herself, at the age of twenty years, with her first child, which was evidently the subject of inherited syphilis. The mother had no knowledge of the disease in herself, but presented characteristic scars about the mouth. There was no evidence that she had suffered from the acquired form of the disease. In the third instance, a woman aged twenty-one years, with Hutchinsonian teeth and scars of old rhagades, brought to the clinic her child of five months, who was affected with inherited syphilis. Six months previously the grandmother had sought advice for a tertiary tuberculo-serpiginous syphilide on the left forearm. The same mother bore a second child five years later, which was also the subject of inherited syphilis. In her case there was likewise no reason for suspecting a reinfection. The father of these two children and the father of the child in the second case could not be examined, but Boeck lays little emphasis upon this, as he considers infection from the father excessively rare, if at all possible. In an experience of thirty years he has met with not a single instance in which the facts have obliged him to admit paternal infection of the offspring, and this view is supported by many other observers. That the mother of the first child had previously borne two healthy infants is nothing anomalous, as the more remote the date of infection of the mother, the more uncertain and variable will be the fate of the offspring. The time between the infection of the mother and the birth of the children is not too long to admit of her giving them the disease, as in the acquired form women may bear syphilitic children twenty years or even thirty-seven years after inoculation.

In this relation it is interesting to note the opinions of JONATHAN HUTCHINSON, which appear on page 977 of the same journal:

"1. I have never had an opportunity for personally investigating a case in which the evidence convinced me that syphilis had been transmitted by inheritance to the third generation. Several very interesting ones have been brought under my notice in which at first the facts seemed conclusive, but in which on further examination they broke

down. For the present I do not believe in the possibility of such transmission.

"2. No doubt can, I think, be reasonably entertained as to the transmission of syphilis by the male parent without direct infection of the mother. This is in England by far the most common form of transmission, for it is very rare that married women have suffered from primary disease. . . ."

SURGERY.

UNDER THE CHARGE OF

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Duodenal Ulcer and its Treatment.—D'ARCY POWER (*British Medical Journal*, December 17, 1904) states that the subject of duodenal ulcer has not yet received adequate attention, though its onset is severe and the sequelæ may be dangerous. It is usually thought to be of rare occurrence, but during the last few years the author has had the opportunity of operating upon 7 cases and observing some others. These cases grouped themselves sharply into two classes: those in which the ulcer had perforated and those in which there was no perforation. The cases which perforated required immediate surgical treatment and were sutured with more or less success. Those which did not perforate were either treated at once on account of hemorrhage or came for treatment many years afterward in consequence of duodenal narrowing due to cicatrization of the ulcer. The non-perforating cases were treated by retrocolic gastrojejunostomy. As all the cases were in males it may be presumed that this condition is more common in this sex than in females. After noting in detail the symptomatology of the two classes of cases the author fully considers the question of diagnosis and states that it should be easy to diagnosticate the cases where perforation has occurred, but in practice it is often found to be a matter of very great difficulty. The symptoms are not characteristic and so the diagnosis is often left in abeyance, in the hope that a few hours' delay will render the signs and symptoms more definite. Such advice is likely to prove fatal, for, instead of making the diagnosis clearer time only renders it more obscure. The slight clues which could be picked up shortly after the onset, are soon masked by the peritonitis which follows. Delay not only allows the peritonitis time to develop, but it permits the extravasated contents of the alimentary canal to gain access to the innermost recesses of the peritoneum, so that a subphrenic, pelvic, or iliac abscess may still further complicate a condition which is well-nigh desperate. A rapidly increasing pulse rate

with acute abdominal pain and but slight objective symptoms is an indication for an exploratory operation. The diagnosis becomes even more obscure without operation, and the case may be mistaken for pneumonia, appendicitis, or peritonitis, due to causes other than intestinal perforation. The lesson to be learned from the cases that have perforated is to operate early. The perforation often takes place without warning, and it thus occurs in persons who are otherwise in excellent health. Such persons bear an abdominal operation very well, and it is better to open an abdomen needlessly than wait until the symptoms of peritonitis make an operation imperative. The diagnosis of the non-perforative cases is even more difficult, for the reason that there is no pathognomonic sign of non-perforating ulcer of the duodenum. After noting in detail the various symptoms the author reaches the following conclusions: (1) Duodenal ulcers are not very uncommon. (2) So far as he has seen them, duodenal ulcers are single and are more common in men than in women. (3) Duodenal ulcers may perforate and cause acute symptoms, or they may heal, and by cicatrization lead to symptoms of chronic duodenal obstruction. (4) The sequelæ of a healed ulcer may be so remote that the symptoms are mistaken for those due to cancer of the pylorus, and the patient is allowed to drift from bad to worse under the erroneous notion that he is bound to die. (5) There is no means of recognizing the existence of a duodenal ulcer in a great many cases until it perforates or the results of its cicatrization become manifest. (6) The treatment of duodenal ulcer consists (*a*) in the direct suture of a perforated ulcer, the prognosis being less favorable than in similar cases of perforation; (*b*) the performance of gastrojejunostomy in cases of dilated stomach due to duodenal constriction, the prognosis being the most favorable of all the conditions for which this operation is performed at the present time.

The Intravenous Injection of Antitoxin in Diphtheria.—BIERNACKI and MUIR (*Lancet*, December 24, 1904) state that they are familiar with the work of Cairns, who has claimed increased efficacy for this method and who has stated that there resulted a quick subsidence of the glandular enlargement, a strikingly rapid decline in the toxæmia, and in pneumonic cases a marked diminution in the restlessness. In 50 cases, 20 of which were treated by this method, there were only 3 deaths, a mortality of only 6 per cent. Of these 17 were tracheotomy cases, with only 1 death, a mortality of only 5.8 per cent. The authors state that they tried the method in 7 cases with 5 deaths in one series, and in another, in 38 cases with 3 deaths.

After reporting these cases in detail they state in conclusion that in attempting to estimate the beneficial effects of antitoxin given intravenously those cases must be discounted in which a marked improvement follows intubation or tracheotomy, since this may be due mainly or entirely to relief of the obstruction. Nevertheless, it will be noted that of 9 cases operated on early, only 1 died, and this must be regarded as a low mortality. Even in cases other than laryngeal, it would seem very difficult if not impossible to say of any individual patient that a better result was obtained than might have followed subcutaneous injection. However, there was a fatality of 3 in 38 selected severe cases. This result seems to be in favor of the intravenous method. At the same time, although many of the cases treated were undoubtedly

very severe, there has been a general fall in the fatality of diphtheria in the district, and this leaves room for speculation as to whether the subcutaneous method might not have yielded better results than in the past.

The Germ Cell Theory of Cancer.—GRUNBAUM (abstract in the *British Medical Journal*, December 17, 1904). The explanation of the genesis of malignant growths has been brought nearer to us by two notable investigations. They are (1) the proof of the morphological continuity of the germ cells, and (2) the discovery of heterotype mitosis in malignant tumors. They corroborate and fortify each other in a remarkable way. Until recently it was always taught that the sexual cells arose in the embryo from a layer of peritoneal epithelium, known, it is true, as the germinal layer, but not otherwise distinguished in origin from the remaining cells of the embryo. It is to the credit of Dr. James Beard to have definitely disproved this method of formation and to have clearly shown the morphological continuity of the germ cells. He has combined with this the suggestive hypothesis of an antithetic alteration of generations also in man.

Put very briefly, this may be stated as follows: After the union of the sperm and the egg, there is formed by the subdivision of the united cell what corresponds to a larva or trophoblast; in man the chorion. From one of its cells there is formed, as it were, a number of spores. One of these spores ultimately becomes the embryo, which includes within itself the remaining spores to become its sexual cells, which are thus not formed from the embryo, but are handed down to it, and for which it merely forms a home.

In passing to their proper position in the embryo these germ cells may go astray, and may in some animals be demonstrated for a time in all sorts of abnormal positions. Dr. Beard was also the first to suggest that the aberrant germ cell might be the origin of cancerous growths, although he now prefers to consider it as an identical twin, namely, an embryo developed, or rather undeveloped, from the same germ cell. The former idea would seem to be the preferable one.

But what proof is there of any connection between a germ cell and a malignant growth? This has been supplied by the discovery of Professor J. B. Farmer with Messrs. Moore and Walker. They found in malignant tumors a form of nuclear division which normally occurs only in the sexual cells, among the spermatozoa and ova. One of the distinguishing features of this heterotype mitosis is the formation by the dividing cells of only half the normal number of chromosomes. Both this and the other characteristics were observed in the cells of malignant growths; there must, therefore, be something in common between the germ cell and the tumor cell.

In innocent tumors they were unable to find this form of nuclear division and considered its absence an essential difference between the two forms of growth. It would seem to corroborate the view that the distinction between malignant and innocent tumors is not fundamental. We know that clinically one form may pass into the other.

The heterotype mitosis does not supervene at once in the evolution of the sexual cells. In the testis it is not before the third division, and in the ovary when the membrana granulosa begins to proliferate, and the discus proligerus is formed; definite proof of their non-somatic

origin. In the same way heterotype mitosis may not occur at first in tumors, and during this stage they are not malignant, but when it supervenes they become so. Why this change should occur is another matter; but it is no more mysterious in the case of a tumor than in that of the testis or ovary.

Given the cell or cells of the potential growth it seems not improbable that the toxin of a parasite, the short stimulus of a trauma, the long-continued stimulus of chronic irritation or the chemical conditions of disordered metabolism, might suffice to let loose their energies and thus one or all of the alleged causes in turn have their share.

The sign-post of further investigation of the problem points along the road of embryological and cytological inquiry.

THERAPEUTICS.

UNDER THE CHARGE OF

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ASSISTED BY

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Hepatic Opotherapy.—DR. JULES REYNAULT reports an instance of cirrhosis of the liver, with splenomegaly, ascites, and œdema of feet which had resisted other modes of treatment, but in which the following mode of procedure achieved a rather remarkable result. The patient was given the following prescription: ox gall simmered over a slow fire until it had reached a syrupy consistency, 5 drachms; powdered rhubarb and powdered cinchona, equal parts, q.s.; make 40 pills. Four of these were taken daily and the following diet was ordered. Each day, calf, pork, or sheep liver, raw or slightly broiled, 4½ ounces; raw spleen of pork or sheep reduced to a pulp, 1½ ounces; milk 2 to 3 quarts. After seven days the patient was much improved and all the symptoms were less marked. The spleen pulp was now stopped because of the nausea which it had produced, the liver was reduced to 3 ounces and the pills to two per day. Eggs were allowed. After three weeks of treatment the special diet was abandoned and a few days later the pills, and the patient returned to work. The amelioration is permanent, the liver and spleen, while not so small as normal have diminished in size and the general condition is excellent.—*Revue de therapeutique*, 1904, No. 20, p. 695.

The Action of Radium in Cutaneous Affections.—DR. SCHOLTZ states that the action of the rays emanating from radium resemble the *x*-rays, but differ in the following points: (1) Radium acts more energetically upon the vessels, its deep action is more marked, and it possesses

bactericidal properties. (2) Radium exercises a more intense and more favorable effect than the *x*-rays upon malignant cutaneous neoplasms (especially carcinomata) and upon small telangiectases and angiomata. (3) Radium may easily be employed within the cavities of the organism. —*Revue therapeutique*, 1904, No. 20, p. 709.

Therapeutic Indications Furnished by Sphygmometer in Enteric Fever.—Drs. Carriere and Dancourt consider that it is necessary to distinguish the cases with hypotension from those with hypertension. Hypertension is likely to precede the onset of complications, congestive or hemorrhagic, depending upon the localization of the hyperæmia. To avoid these complications we should combat the excess of arterial tension. This may be done by means of the vasodilators and in cases of heart weakness by these in connection with heart stimulants. One may use amyl nitrite, the other nitrites or trinitrin, but the author gives preference to spiritus ætheris nitrosi, of which he gives 20 to 100 drops during the twenty-four hours. In elevating arterial tension we may use agents which act upon the peripheral vessels by vasoconstriction or those which stimulate the heart's action. In enteric fever the vasoconstrictors are dangerous because of the increased work which they impose upon a previously weakened heart. The authors reject the vasoconstrictors, choosing instead cardiac stimulants, of which digitalis, caffeine, and sparteine are the most effective. They usually prescribe the first in the form of the infusion. If intolerance occurs, caffeine is substituted.—*Revue de médecine*, 1904, Nos. 7 and 8, pp. 537 and 643.

Urotropin, Methylene Citric Acid and Urotropin Methylene Citrate.—Dr. Arthur Nicolaier records his exhaustive studies of these drugs, confirming his statement made in 1895 that urotropin in 7½ grain doses 2 or 3 times a day in ½ pint of water is harmless and soon after administration appears in the urine, that the urine, without losing its acidity acquires uric acid solvent properties and an inhibitory action on bacterial growth except upon that of the tubercle bacillus. At times there occurs non-tuberculous bacterial infection in which the desired effect of urotropin is not obtained; consequently experiments were made with helmitol (urotropin methylene-citrate or new-urotropin). First, experiments were made with methylene citric acid from which it was concluded that this drug did not give off formaldehyde in the urine as does urotropin and may produce skin eruptions, and digestive disturbances. These same objections hold good in regard to citarin, the sodium salt of methylene citric acid. With regard to helmitol it was proven that in doses which are the coefficient of the usual urotropin doses—*i. e.*, which are two and one-half times as great as the latter, helmitol is effective in exactly the same conditions in which urotropin is effective and ineffective in exactly the same cases in which urotropin is ineffective. It has also been shown both bacteriologically and clinically to be dependent for its therapeutic efficiency upon its urotropin content and by reason of the contained methylene citric acid to be capable of causing unpleasant by-effects.—*Deutsche Archiv für klinische Medizin*, 1904, Nos. 1 and 2, p. 181.

Thiol in Dermatology.—M. Leredde considers that this substance, which is derived from oil of wood-tar and occurs in either powdered

or liquid form, is useful in dermatological practice, where it may be employed in solutions, ointments, glyceroles, etc., of from 5 to 10 per cent. strength. Its chief indications are in eczema and other pruriginous affections. In the acute type of the former condition thiol is useful in the declining stages. In chronic eczema it is also useful in combination with other substances. Its antipruritic action is excellent, and it may be employed in acute pruritus, prurigo, licheniform lesions, etc. Thiol combined with other antiparasitics is useful in the seborrhœides, intertrigo, psoriasis, artificial dermatites, and it acts well in the acute dermatoses of infants. No bad effects are attendant upon its use, and it is worthy, according to the author, of more general employment.—*La presse médicale*, 1904, No. 91, p. 727.

Thigenol in Gynecology.—Dr. Raoul Blondel has employed this agent for a year, both in dispensary and in private practice, and considers it a very valuable one. Its action is that of ichthyol, but it is to be preferred to this substance, since it is more effective and has none of the disadvantages of the latter, either in regard to odor or to absorption and consequent toxic action. Employed on tampons thigenol is indicated in subacute inflammatory uterine conditions. The large, congested uterus diminishes in size under its application, the hemorrhages cease, and normal menstrual function becomes established. Perhaps the best effects of this drug are achieved in inflammations of the adnexa, parametritis and perimetritis. The author uses in these conditions tampons of equal parts of thigenol and glycerin and states that rapid cessation of the pain and absorption of the exudate result. Thigenol is valuable also in gonorrhœal inflammations of the urethra and paraurethral glands. Here it may be used pure on small tampons of cotton inserted just after micturition or it may be introduced into the glands through a platinum needle.—*Revue de thérapeutique*, 1904, No. 24, p. 834.

Antiseptic Treatment of Middle-ear Suppuration.—Dr. Heinrich Neumann considers that the oxygen liberated by hydrogen dioxide will destroy the anaërobic micro-organisms which exist in old suppurative conditions, but will not affect aërobic bacteria in the same way. Consequently he combines hydrogen dioxide with potassium permanganate. The combination possesses not only the catalytic power of the latter substance, but also its coagulating and oxygenating effect. The mixture gives off a large amount of oxygen and the evolution takes place slowly; consequently its action on the bacteria is prolonged. In tympanic perforations of large size the author proceeds as follows: The canal is cleansed by antiseptic irrigations and then filled with a warm solution of potassium permanganate (1 per cent.). This is allowed to remain for a few minutes, is then removed and the canal is filled with 3 per cent. hydrogen dioxide, which is permitted to exert its action for six to eight minutes. This treatment has achieved remarkable result in otorrhœas of obstinate type.—*Wiener medizinische Presse*, 1904, No. 46, p. 2181.

Radium in Joint Affections.—M. Soupault has obtained excellent results from the emanations of radium in subacute joint affections. He reports patients affected with double hydrarthrosis, subacute gono-

coccal arthritis of wrist and hand, subacute articular rheumatism and subacute gout due to plumbism as cured by this means. In acute cases the author met with no success with the radium.—*La presse médicale*, 1904, No. 92, p. 734.

Neuronal.—Chemically this hypnotic is bromodiethylacetamide, a white, crystalline powder slightly soluble in water, easily soluble in alcohol, and resembling menthol in taste, with the addition of slight bitterness. According to Siebert, Becker, and others it produces a quiet slumber about one-half hour after its administration, with no unpleasant after-effects. The drug seems to have no cumulative action and its dosage in mild insomnia is 7½ grains, in more obstinate cases from 20 to 30 grains.—*Therapeutische Monatshefte*, 1904, No. 11, p. 591.

The Action of Digitalis in Combination with Caffeine.—Dr. Ludwig Braun states that the simultaneous administration of digitalis and caffeine has an especially good effect in mitral stenosis, in cardiac disease with symptoms of heart weakness, in aortic lesions, and in arteriosclerosis complicated with aortic insufficiency. The author has studied the effects of these drugs, both separately and in combination, upon the heart and coronary arteries of mammals, and believes that when they are given simultaneously the vasoconstrictor effect of the digitalis is not produced and the action of the heart is favorably influenced. Consequently the favorable action of the combination is attributed to the improvement produced in the coronary circulation. In effect, the only disadvantage of digitalis, its vasoconstrictor action, is obviated by the combination with caffeine, for, in spite of the increase in arterial pressure produced by the digitalis, the coronary arteries remain widely open, and the conditions for plentiful nutrition of the heart remain favorable. The experiments upon animals are borne out by clinical experience, which proves that the effects of the combination of digitalis and caffeine are not produced by either drug alone.—*La semaine médicale*, 1904, No. 47, p. 383.

Pyrogallic Acid in Cystitis.—M. F. Minet reports good results from the use of this agent in tuberculous cystitis. His observations are based on 60 cases, and he recommends the drug in all forms of this condition, both primary and secondary, except that attended by hemorrhage. The technique is simple, and consists in the instillation of 75 drops of a 1:50 to 1:20 aqueous solution. If retention exists the bladder should first be emptied. Local treatment, according to the author, is less necessary in young patients who are able to take a prolonged rest and fresh-air cure.—*Le progrès médicale*, 1904, No. 47, p. 420.

Hydrotherapy and Tetanus.—Dr. J. Sadger discusses a method of hydrotherapy in tetanus simpler and easier to carry out without the help of assistants than the Preissnitz process. The four steps in his procedure are as follows: (1) a dry pack until sweating sets in; (2) a cool bath with pouring of cold water over the patient, especially over the spine; (3) a wet pack, and (4) the process described under (2) repeated. He cites an instance in which this treatment was used

successfully where the symptoms, including a relapse, were so severe as to lead one to expect a fatal outcome. Other authorities have called attention to the value of cold water in this disease, which is allowed to fall from a height upon the patient. But the commoner methods have consisted in the use of heat. However, by combining both these agents one obtains a favorable result in the condition of the nervous system and in increased perspiration. Also one finds improvement in the patient's general condition, in the relief of cramps, and in the elimination of the toxins.—*Zentralblatt f. die gesammte Therapie*, 1904, No. 11, p. 565.

OBSTETRICS.

UNDER THE CHARGE OF

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Spontaneous Rupture of the Uterus during Pregnancy through the Scar of the Cæsarean Wound.—KERR (*Journal of Obstetrics and Gynecology of the British Empire*, November, 1904) reports the case of a patient in her fourth pregnancy, on whom he had two years previously performed Cæsarean section. The first two pregnancies had been terminated by the birth of dead children, and the pelvis was contracted. Cæsarean section was done, and the fundal incision of Fritsch was employed. The child weighed eight pounds, was readily extracted, and the uterine wound closed with little trouble. The patient made a good recovery.

She returned to the hospital in the thirty-seventh week of her fourth pregnancy for a second section. She was in good condition, the abdomen irregularly enlarged, the greater swelling on the right side. The scar of the previous operation was firm, fetal movements were active, the heart sounds not heard, and the cervix admitted one finger, but was not shortened. On admission the patient received an enema and had some abdominal discomfort. She, however, slept. In the early morning a bloody discharge from the vagina was noticed, with slight pain in the right iliac region. The pulse was normal and the temperature slightly below the average. There was no sickness or vomiting, but pain had spread over the entire abdomen. The abdomen gradually became tender; although the pulse remained good until after several hours, the temperature was subnormal, the breathing rapid, the abdomen tender, and the pulse 90. On placing the hand above the abdomen, the fœtus could be felt with abnormal plainness. Two tumors could be distinguished, and at the sides of the abdomen there was slight dulness on percussion. On vaginal examination no presenting part could be felt, but the finger was blood-stained.

When the abdomen was opened a large quantity of dark blood escaped

and the membranes and placenta were found intact. The uterus was retracted behind and downward toward the pelvis. The membranes were opened and a fully developed dead child removed. On examination a transverse rupture of the uterus through the scar of the previous incision was present. Supravaginal hysterectomy was performed, and the patient made a good recovery.

In diagnosticating the rupture the symptoms were so slight that it was thought that the rupture was incomplete and limited by adhesions. At operation it was found that there were but two slight adhesions, one to the omentum and one to the abdominal wall.

Kerr believes that a scar through the fundus is more apt to give way than the ordinary longitudinal one. The anterior incision is better. On one occasion, on performing a second operation, he found the old cicatrix at the fundus much thinner than other portions of the uterus.

Another feature which predisposed to rupture of the scar was the fact that at the first operation the placenta had been attached at this point. Further, in the second pregnancy the placenta was situated over the scar.

As regards the result of the operation in general, Kerr states that he has now had 30 cases with 2 deaths, a mortality of 6.6 per cent. He has had two repeated Cæsarean sections with good results. He adds the report of 5 cases in which the scar ruptured in a pregnancy following that terminated by Cæsarean section.

Two Cæsarean Sections for Eclampsia.—In the *Zentralblatt für Gynäkologie*, 1904, No. 45, WANNER reports 2 cases of Cæsarean section for eclampsia. In both patients the convulsions were exceedingly severe. The patients were from eight to fourteen days before term, and there was no sign of uterine contractions.

The first patient was a primipara with almost total suppression of urine, rapid pulse, and temperature slightly raised. The cervix was not softened or dilated. Abdominal Cæsarean section permitted the delivery of a living, well-developed male child. The patient died forty-eight hours after operation, with œdema of the lungs.

The second case was that of a primipara, greatly asphyxiated, with rapid pulse and profoundly comatose. The head was freely movable above the pelvic brim, the cervix tightly closed, and there were no uterine contractions. Vaginal Cæsarean section was performed by making an incision through the vulva and perineum on the right side, opening the cervix with scissors upon one side, and then splitting the cervix on the anterior and posterior wall to the internal os. Version was performed and a living, vigorous child extracted. The placenta was removed by expression with free bleeding. The uterus was pressed down, the cervix seized with forceps, and the incisions closed with catgut. After delivery the patient had one convulsion. Four hours after delivery there was hemorrhage, which rendered tamponing of the uterus necessary. The patient also received the injection of salines. On the following day the tampons were removed and the incision in the perineum and pelvic floor closed. The patient remained partly comatose for three days, but finally recovered.

In the vaginal operation incision through the pelvic floor and vulva was made to avoid the necessity of pushing back the bladder from the anterior uterine wall.

Carcinoma of the Cervix Complicating Pregnancy and Labor.—In the *British Medical Journal*, November 12, 1904, KERR describes 2 cases of carcinoma of the cervix complicating pregnancy. He states that in the Glasgow Maternity Hospital, they have had, in addition, 4 inoperable cases during the last ten years.

The first case was a multipara, whose first symptom of malignant disease was severe hemorrhage. When seen she was far advanced in pregnancy, and the carcinoma had involved the entire cervix. When admitted to the hospital, no enlargement of the lymphatic glands could be made out. The pregnancy was nearly at term; the heart and lungs were normal. The patient was delivered by Cæsarean section, and a living healthy male child was extracted. The uterus was opened by incision across the fundus to facilitate its complete removal. After emptying the uterus the operator removed it down to the cervix, but the cervix and surrounding tissues were so involved that its removal was impossible. The stump was closed by layers of peritoneum. The patient made a good recovery from the operation, leaving the hospital thirty-three days afterward. She died six weeks after returning home.

The second case was also a multipara in the thirty-eighth week of gestation. Masses were felt upon both lips of the cervix in this case, and there was partial dilatation. Uterine contractions came on and operation was performed.

The cervix was first amputated through the vagina and both uterine arteries were tied. The vagina was then thoroughly cleansed, the hands again sterilized, and Cæsarean section performed by abdominal incision. The child was removed, slightly asphyxiated, but revived. The remainder of the uterus was removed through the abdomen, and the vaginal vault was closed. The patient died on the fifth day after operation, from septic peritonitis.

These cases are types of the two general classes into which they may be divided; first are the inoperable cases, in which Cæsarean section was performed to save the life of the child without the attempt to completely remove the cancer; the second was an example of an operable case, and illustrates the dangers of the operation at the time when pregnancy has nearly reached full term. In dealing with inoperable cases, it is of the greatest importance that Cæsarean section should be performed before labor has begun. The uterus, to prevent infection, should be removed, and the stump treated by careful closure of the peritoneum to prevent infection. In operable cases the uterus may be removed through the vagina as late as the fourth month of gestation. After that the most satisfactory method consists in performing Cæsarean section, after which supravaginal amputation is practised, followed by the removal of the cervix through the vagina. This avoids the risk of contaminating the peritoneum by bringing the cancer through the abdomen. By downward traction the malignant growth is removed from below.

Lumbar Puncture in Eclampsia.—In the *Zentralblatt für Gynäkologie*, 1904, No. 45, HENKEL gives the results of 16 cases of eclampsia in Ohlshausen's clinic in Berlin, in which lumbar puncture with extraction of cerebrospinal fluid was performed. These cases illustrated well different types of eclampsia. Some occurred in primiparæ, others in multiparæ soon after labor, and some in the early stages or very beginning of labor. In a considerable number lumbar puncture was per-

formed and fluid extracted, followed by the injection of cocaine and scopolamine.

In some cases the quantity of cerebrospinal fluid was found increased; in others diminished. In some there seemed to be no fluid present, and in others the quantity of fluid seemed normal. The mortality was 25 per cent. This is no improvement on the average mortality of eclampsia under hospital treatment.

So far as these experiments give information concerning the increase of cerebrospinal fluid in eclampsia, in 7 of these patients the spinal canal was empty, and 1 of these patients died; in 5 cases very little cerebrospinal fluid escaped, and 2 of these patients died. In 4 cases the quantity of cerebrospinal fluid was greatly increased, and 1 of these cases died. From this series of cases no practical result whatever followed lumbar puncture and the use of cocaine and scopolamine by injection.

KLEINWÄCHTER (*Zentralblatt für Gynäkologie*, 1904, No. 45) reports a case of eclampsia in which lumbar puncture was employed. The patient had fifteen convulsions, was deeply comatose and greatly prostrated. She had been given chloral hydrate, diaphoretics, thyroid extract, croton oil, and injections of saline fluid into the bowel. Salt solution was also injected beneath the skin and lumbar puncture was employed. One and a half drachms of cerebrospinal fluid was allowed to escape. The fluid escaped in a small stream and not in drops. Cocaine was injected, and salt solution was also introduced into the bowel. The patient improved and was subsequently treated by thyroid extract, sulphate of magnesium, and iron. She ultimately recovered.

GYNECOLOGY.

UNDER THE CHARGE OF

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Endometritis Following Gonorrhœal Vulvovaginitis in Children.—JUNG (*Zentralblatt für Gynäkologie*, No. 33, 1904) concludes a clinical study of this subject with the following statements: 1. In most cases of vulvovaginitis careful examination of the cervical secretion is usually sufficient. 2. The removal of mucus from the portio on a swab is not enough for diagnostic purposes. 3. The writer's observations, as well as those of Gassmann and Buscke, prove that while gonorrhœal endometritis does occur as a complication of infantile vulvovaginitis, it is infrequent (10 per cent.), while disease of the annexa is very rare. 4. The cervical secretion should be examined bacteriologically in every case of vaginitis in children, especially when it is of long standing. 5. It is doubtful if it is advisable to apply the treatment directly to the cervical canal.

Styptol in Uterine Hemorrhage.—WITTBAUER (*Zentralblatt für Gynäkologie*, No. 33, 1904) has used this drug in a number of cases, and concludes that its action is similar to that of stypticin in cases of menorrhagia, and especially in climacteric bleeding, though it is apparently more effective. It does not take the place of ergotin, however, in cases in which it is desirable to cause uterine contractions. Styptol is harmless and is much cheaper than stypticin, and is therefore applicable to hospital and dispensary use.

Microscopic Diagnosis of Abortion.—HITSCHMANN (*Zentralblatt für Gynäkologie*, No. 32, 1904), referring to Opitz's conclusions that characteristic glands are always found in tissue removed from the pregnant uterus, describes the same typical glands which he found in scrapings from non-gravid uteri. These are distinguished by the presence of small papillary projections into the lumen of the gland, covered with epithelium, an appearance also noted in the writer's specimens. He concludes that the swelling of the uterine mucosa during menstruation may cause the same peculiar change in the glands as nidation.

Tubal Menstruation.—THORN (*Zentralblatt für Gynäkologie*, No. 32, 1904), after discussing this subject at some length and citing two cases from his own practice, arrives at the conclusion that there may occur rarely a sort of vicarious hemorrhage from the tubal mucous membrane. In both of his cases the tubes were diseased, and he holds that this was probably true in every instance in which this phenomenon was noted. At any rate, he denies the occurrence of a normal tubal menstruation as claimed by some authors. Doubtless the healthy tube shares in the general menstrual congestion, but no more.

Premature Menopause.—SIREDY (*Zentralblatt für Gynäkologie*, No. 32, 1904) reports five cases in which climacteric symptoms were wanting. In all menstruation began late and was always scanty. The writer attributed the condition to imperfect development of the sexual organs, as evidenced by atrophy of the cervix uteri.

Hartmann (*ibid.*) reports a similar case in a healthy girl, aged nineteen years, whose periods had been regular for four years.

Etiology of Kraurosis Vulvæ.—JUNG (*Zeitschrift für Geb. und Gyn.*, Band lii., Heft 1) believes, from the study of several cases, that kraurosis is only an advanced stage of chronic vulvitis, since there is no difference in their macroscopic appearance or clinical symptoms. The histological difference is quantitative rather than qualitative. Kraurosis is not to be regarded as a separate disease.

Parotitis Following Curettement.—CONDAMIN (*Lyon méd.*, April 12, 1904) reports a case of parotitis after aseptic curettement, and infers that this complication of gynecological operations is not necessarily of infectious origin, but may be due to reflex nerve irritation through the medium of the sympathetic and cervical ganglia. Thirst after laparotomy may be referred to the same cause—diminution or suspension of the salivary secretion. The invasion of the glands by microbes from the mouth may also play a part; hence the writer

recommends the use of a mouth-wash for a few days after operation, or even the administration of pilocarpine.

Malignant Degeneration of Ovarian Cysts.—BARNSBY (*Annales de gyn. et d'obstétrique*, No. 8, 1904) found malignant degeneration in 36 per cent. of his cases. He believes that ovarian cystomata are nearly always benign at first, the cancerous condition being secondary. In 32 operations only one patient died. Recurrence took place in 6 cases, in 5 of which rupture of the cyst occurred spontaneously or during operation. The writer thinks that a cyst which is suspected to be cancerous should be removed entire without previous puncture.

Shortening of the Round Ligaments.—DOLERIS (*La Gynécologie*, August, 1904) concludes an article on this subject as follows: 1. Fixation of the uterus is an unphysiological operation, depending for its success on pathological conditions; indirect, or ligamentary, hysteropexy is always preferable. 2. Operations for fixation of the organ are not unattended with risk and the ultimate results are uncertain. 3. From an obstetrical point of view extraperitoneal or intraperitoneal shortening of the round ligaments is much safer, as shown in 177 cases collected by the writer in which there was no instance of dystocia. 4. These operations meet all indications, whether the case is simple or complicated. 5. It is not enough to retain the uterus in its normal position; plastic operations on the cervix and pelvic floor must not be omitted when indicated.

Foreign Body in the Vagina.—ORLOY (*Roussky Wratsch; La Gynécologie*, August, 1904) reports the case of a peasant woman, aged sixty-six years, who entered the hospital with pain in the lower abdomen and a foul discharge. She stated that thirty years before, after the birth of her child, she had procidentia, which was aggravated after a second confinement. In order to keep the uterus in position she had introduced into the vagina a croquet ball, which gave entire relief and had not been removed since. On examination the ball was found to be so firmly impacted, on account of the senile atrophy of the tissues, that it was necessary to remove it piecemeal. A few areas of ulcerations were present, which healed in a few days.

Gonorrhœal Inflammation of Skene's Glands.—POLLAK (*Zentralblatt für Gynäkologie*, No. 9, 1904) found the glands involved in 45 out of 100 cases of subacute and chronic gonorrhœa in which the diagnosis was confirmed by bacteriological examinations. In 31 cases only one gland was involved. The writer concluded that there was no relation between the number of gonococci found in the urethra and those in the glands.

Paralysis after Narcosis.—GLITSCH (*Zentralblatt für Gynäkologie*, 1904, No. 39) thinks that this accident is due to several causes, viz.: 1. Forced elevation of the arms and simultaneous rotation outward, causing pressure of the clavicle against the first rib and consequent involvement of the brachial plexus. 2. When the capsule of the shoul-

der-joint is opened in the cadaver, so that the head of the humerus can slip forward, this pressure is absent. 3. Under the same conditions with the arm raised above the horizontal, the head of the humerus presses on the plexus.

The writer infers that clinically postoperative paralysis results from temporary pressure of the head on the infraclavicular portion of the plexus, due to hyperelevation of the arm, with rotation inward. He accordingly favors Holst's method of securing the arms at the sides, resting on cushions. Attention is called to the medico-legal importance of the lesion.

Benign Chorioepithelioma.—VELITS (*Zeitschrift für Geb. u. Gyn.*, Band lii., Heft 2) describes the case of a multipara, aged thirty-four years, who, a month after the excision of a vesicular mole, began to have uterine hemorrhages. A growth was removed, which was pronounced to be chorioepithelioma. The uterus was extirpated and the patient had remained well for a year.

From a study of this one and other specimens the writer concludes that spontaneous cure of these neoplasms may occur through necrobosis, as shown by the lowered vitality and disappearance of the cells of Langhans and the appearance of wandering cells, which show the separation of the syncytium.

HAMMERSCHLAG (*ibid.*) reports only 4 cases of chorioepithelioma from the Königsberg clinic among 5000 gynecological cases, to which he adds a fifth. The patient's ages ranged from thirty-two to forty-seven years, and all had borne at least seven children. The neoplasm developed each time after a previous abortion; three after molar pregnancy, the time varying from five weeks to two and a quarter years. Three died, two being cured by vaginal extirpation. The ovaries were all diseased, usually showing cystic degeneration.

Physiological Studies of the Uterus.—FRANZ (*Zentralblatt für Gynäkologie*, 1904, No. 24) presents the following deductions regarding the uterine muscle, based on a series of experiments with rabbits as well as with human uteri examined immediately after removal: 1. In rabbits the uterus can always be excited to contract by mechanical or electric stimulation. 2. When drawn out of the abdominal cavity in the living animal and suspended it contracts spontaneously, possibly from a lowering of the temperature. 3. The virgin organ is less susceptible to stimulation than the nulliparous, and the pregnant most of all. 4. When removed from the body the uterus in the rabbit responds but slightly to electric stimulation, even when freshly excised. 5. The organ responds most vigorously to thermic stimulation (immersion in salt solution at a temperature of from 50° to 60° C.), but the muscle soon becomes relaxed. Low temperatures (0° to 5° C.) cause contractions, which increase gradually and persist for a long time. 6. A series of rhythmic contractions can be produced by electric stimulation. 7. There is no marked difference between the muscular irritability of the freshly excised uterus in the rabbit and human female, except that there is a weaker response in the latter to electric reaction, thermic stimulation having the most marked effect, even in atrophic post-climacteric uteri. 8. The freshly excised vagina in the rabbit shows a similar response to

stimuli, especially to thermic, when the contractions become rhythmical. 9. Chloral hydrate prevent the contractions in a uterus suspended *in situ*, ergotin increases the muscular irritability and tendency to rhythmical contractions, atropin has a negative effect, while nicotin produces strong tetanic contraction.

OTOLOGY.

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A Contribution to the Pathology and Pathological Anatomy of Congenital Deaf-mutism.—G. ALEXANDER (*Archiv. f. Ohrenheilkunde* Band lxi., Heft 3) reports the pathology of an interesting case of congenital deaf-mutism. The deaf-mute, who, for a long time, had been an inmate of a poor-house in his native town in lower Austria, came to Vienna and sought work. He was employed variously and had no symptoms of any disturbance of equilibrium until finally, being engaged on work on a scaffolding a few metres high, he fell and suffered an injury to his urethra (rupture of the urethra). This injury was followed by pyæmia, of which he died. The organs of hearing were obtained and showed the following pathological conditions:

1. The static labyrinth: The nerve branches and the vestibular ganglia were atrophied, and the nerve terminations showed a corresponding degree of atrophy. The neuroepithelium was found to be lower than normal and showed an evident diminution in the number of sense cells and a relative increase in the number of supporting cells. The cupulæ in the ampullæ were smaller in size than the normal and, in utriculus and sacculus, the otolith membranes and the otoliths were also smaller. The epithelial lining of the static labyrinth, aside from the neuroepithelium, was, for the most part, normal, but here and there there were diffuse or circumscribed thickenings. In places there were epithelium-lined cysts arising from the epithelial layer and projecting into either the endolymphatic or perilymphatic lumina. The whole static labyrinth and its connective nerve branches and ganglia were markedly anæmic and free from pigment. The ductus endolymphaticus, the utriculosaccular canal, and the ductus reuniens were normal. The bony capsule of the static labyrinth was normal.

2. The acoustic labyrinth: The cochlear nerve presented in its entire course an evident atrophy (about one-third of the normal), and its ganglion showed a corresponding atrophy. The grade of atrophy (hypoplasia) was not the same in all places, but varied with that found in the corresponding level of the peripheral endings. The spiral ganglion was found very different from the normal (a spiral course

being evident only at the beginning and in the basal turn). From the end of the basal turn upward the ganglion was represented only by a heap of cells which was situated in the axial part of the cochlea. From this it resulted that the peripheral nerve fibres of the middle and apical turns of the cochlea were much longer than the normal.

The osseous cochlea, considered in its bearings on the pathology of congenital deafness, presented a most interesting condition. While the outer wall, except for the fact that the end of the middle turn and the apical were a little decreased in height, was normal, the whole bony tissue of the modiolus and the lamina spiralis ossea, from the level of the middle turn upward, was wanting. Likewise, in the vestibular division and in the basal turn, the lamina spiralis ossea secundaria was wanting. This failure of the bony modiolus had as a result in the region above the beginning of the middle turn that the scala vestibuli of one turn formed with the scala tympani of the next turn a common space. The helicotrema was wanting and the membranous apical turn closed off the cupola completely below. The ganglion found in the cochlea and its peripheral fibres were quite surrounded in a mass of connective tissue, and in place of the lamina spiralis ossea there were fibrous plates between which ran the atrophic nerve branches.

The membrana basilaris was found in some places particularly thick, in others, thinned. The epithelium on the surface of the membrane directed toward the tympanic scala was, for the most part, present. The spiral vessel was lacking in many places entirely. The crista spiralis was for the most part flattened out and its substantia propria was lacking. Here and there in the crista were cysts lined with epithelium and containing a homogeneous substance. The membrane of Corti was, in the altered middle and apical turns of the cochlea, in its normal position, but in places it was found in the sulcus spiralis internus, rolled up and surrounded by an epithelial border. The epithelium, which here surrounded it was continuous with that of the sulcus spiralis internus. The papilla basilaris was absolutely normal in no situation. In the lower part it was entirely wanting. In other parts, *e. g.*, in the upper turns, it was evidently in an embryological stage, consisting of several rows of epithelium, showing neither differentiated hair cells nor rods. However, many of the cells presented projecting hairs which were in contact with the altered membrane of Corti. In places, the rods of Corti were present intact, and in others, in a rudimentary condition. In the papilla itself, the typical hair cells were wanting throughout. The ligamentum spirale showed, where the membranous canal was preserved, the normal form. In its structure it was in places of a richer cell structure, and in others, the cell elements were few in number. The stria vascularis showed a varied condition. In places entirely wanting, in others it was replaced with pavement epithelium alternating with cylindrical epithelium. The connective tissue of the stria was sharply divided from the epithelium, showing nowhere the characteristic intimate union. The vas prominens was only in a few places evident, and, indeed, the whole cochlea was poorly supplied with bloodvessels.

Alexander regards the condition of the cochlea as evidently a result of arrested embryological development, and finds that an analogous condition obtains in the human embryo at the age of two to three months. As to the condition in the static labyrinth, Alexander has found a

similar condition in the static labyrinth of the dancing-mouse. The dancing-mouse has been found, in experiments conducted by Kreidl and Alexander, to be unable to walk on a narrow support for any distance, although, in ordinary walking, it shows no disturbance of equilibrium. He, therefore, suggests that here in this case we have a physiological analogy. In ordinary occupations, this deaf-mute showed no disturbance of equilibrium. Only when engaged on work on a narrow staging did this faulty equilibration become evident, leading to the deaf-mute's fall and injury which was followed by his death.

Epileptiform Convulsions in a Case of Chronic Suppurative Middle-ear Disease; Recovery Following Mastoid Operation and Lumbar Puncture.—HUBER (*Annals of Otology*, April, 1904) presents the following case:

The patient, a child aged two and a half years, had a history of adenoids and of a fetid discharge, from the right ear, of two years' duration.

For a period of two weeks preceding examination the child had been restless, crying a great deal, and finally, shortly before the date of the examination, had a convulsive attack lasting from two to three minutes; these attacks were repeated, sometimes at half-hour intervals, occasionally accompanied by vomiting, and occurred impartially by day or by night, apparently without extraneous exciting cause.

On examination the child was apathetic, in a half comatose condition; the extremities were cold and cyanotic; there was convergent strabismus, slight horizontal nystagmus, dilated pupils, the right pupil especially; tâche cérébrale; a pulse of 130, but regular; a temperature of 100°, and exaggerated reflexes.

In the right ear there was a large perforation of the drumhead, through which flowed a copious stream of offensive pus.

Passive movements excited general muscular contraction, and a lumbar puncture was done, with the result of a cessation of this reaction.

The postaural operation revealed a pus cavity with bulging dura, and, on two successive days after operation, the lumbar puncture was repeated and followed by a diminution and then a disappearance of the general symptoms.

On the twenty-second day after operation the child was apparently well and was discharged from treatment.

Bone Conduction and the Sound-transmitting Apparatus of the Middle Ear.—In this continuation of his series of experimental investigations BEZOLD (*Zeitschrift f. Ohrenheilkunde*, Band xlviii.) comes to the conclusion that, with both ears firmly closed, while sound waves conveyed through the medium of the air are not perceived, and while, on the contrary, sound waves conveyed to the head by direct contact with the sounding body are distinctly heard, by bone conduction, in all probability only such sound waves are perceived, in the latter instance, as pass to the percipient organ through the medium of the sound-transmitting apparatus, and such, moreover, as set the sound-transmitting apparatus, and the column of intralabyrinthine fluid equally, in transverse vibration.

The author further concludes, from theoretical deduction based upon his observations, that it is only the transverse translation of the to-and-

fro movements of the membrana basilaris, by its sympathetic vibrating mechanism, that actuates the cells of the organ of Corti, and that the role, in the function of hearing, of the sound-transmitting apparatus consists in the conversion of the longitudinal waves, transmitted through the air or conveyed by bone conduction, into transverse vibrations.

Diseases of the Ear in the Italian, French, German, and Austro-Hungarian Armies.—G. Ostino (*Centralblatt für Ohrenheilkunde*, ii., 8) gives a general review of this subject, based upon army statistics, which must of necessity be subject to difficulties because of lack of uniformity in records as well as differences in rules determining leave of absence and discharge from service; but the mass of material at command gives the statistical review a value for purposes of comparison with similar statistics in civil life.

The exclusion from service of from 1.5 per cent. to 2.4 per cent. of recruits on preliminary physical examination, on account of active diseases of the ear or because of resulting impairment of hearing, serves to decrease the percentage of soldiers who would otherwise be suspended or discharged from service on the same account, and tends to explain the low percentages given.

A comparative study of these statistics is also valuable as throwing light upon the differences in the limitation of the medical examination, as, for instance, in the year 1897 the number of men discharged from service in the Italian army was 1.5 per cent.; the French, 0.93 per cent.; the Prussian, 1.85 per cent., and the Austro-Hungarian, 4 per cent., the reason for the larger last-named percentage being the fact that both monaural deafness and the existence of a dry perforation of the drumhead are sufficient considered reasons for rejection or for discharge from the Austro-Hungarian service.

This question of the influence of a dry perforation upon the estimation of serviceableness has long been a matter of controversy among military surgeons. Eichbaum (*Deutsche Militararztliche Zeitschrift*, 1889) is of the opinion that cases of this kind may be admitted to or retained in service, the causative suppurative process having ceased; unless the perforation is large, half or more of the drumhead; unless there is perforation of the membrane of Shrapnell; unless there are granulations in the middle ear, or if there is also chronic nasopharyngeal catarrh or constitutional disease. Schwartz (*ibid.*, 1900) attaches no importance to the size of the perforation, but considers especially the cause of the aural disease, its duration, the general condition of the patient, and whether both ears are affected or only one.

Scholze (*ibid.*, 1901) is of Hartmann's opinion, that if, after a suppurative cause, there is a persistent opening in the drumhead, with the mucous membrane of the tympanic cavity dry and dermoid in character, there is but slight danger of recurrence, and that such cases are not only admissible to service, but should be made the subjects of treatment in view of the considerable possibility of further bettering their condition. Villaret (*ibid.*, 1899, 1900), as the result of consecutive statistics, proved a considerable increase of ear disease among the soldiers during the months when they practised swimming, and commented critically upon the order that "persistent perforation of the drumhead is no longer to be regarded as reason for rejection or discharge."

Of 321 cases of this kind in the Austro-Hungarian army, 23 only were subsequently admitted to the hospital on account of a recurrence of the suppurative process, the remaining 298 finishing their service without going upon the sick list, although 26 out of that number had some degree of suppuration in the middle ear, these statistics differing considerably from those of Scholze, who found at the military station in Mainz 20.7 per cent., and at the station in Muhlhausen 22.7 per cent. of recurrences of suppurative middle-ear disease.

To the value of statistical research uniformity of statement is most important, and that this does not pertain in the statistical study in question is evident when the different forms of aural disease are taken into consideration, for in the Italian and French statistics no distinction as to the part of the ear affected is made, while the German statistics differentiate between "diseases of the outer ear, including the drum-head," and "diseases of the middle and inner ears, including deafness. The percentage of relationship cases under these two headings in a period of fifteen years—1874 to 1889—was 74.1 per cent. of the former and 25.9 per cent. of the latter, the preponderance of the former being due not only to the greater liability to injuries of the external ear in military service, but also to the fact of the classification of the drum-head as a part of the external, when, properly, it belongs to the middle ear.

Comparative statistical conclusions are rendered difficult, also, not only by the difference in construction of terms in the different services, but by the changes in the quality of material at different times with the influx of new recruits and the changes of station.

As concerns the period of the year in which aural diseases especially prevail, this is influenced by the increase in the number of new recruits as well as by climatic change. For instance, in the Italian army the percentage of ear diseases is higher in March and April, because of the prevalence among the troops of measles and influenza, and higher in January, because of climatic conditions and the influx of cavalry recruits. In the Austro-Hungarian army the percentage increase occurred in the same months for nearly the same reasons, while in the Prussian army the major percentage occurred in July—a circumstance which leads Villaret to infer that this increase is due to the prevalence of swimming and to the effects of the spring field manœuvres, a view scouted by Scholze, who draws attention to the fact that the percentage was less by 0.03 in the three summer than in the three winter months, and that it may, therefore, be not unjustly concluded that the weather had a more prejudicial influence upon the ears of the troops than either swimming, the detonation of fire-arms, or the inhalation of the dust of country roads.

As regards the liability of the different branches of the service, that which was true of the Italian army—a greater percentage of ear disease among the infantry—was measurably true of the other armies here considered, but the French statistics show a very considerable preponderance of ear diseases, as high as 5.9 per cent. among the troops on foreign service and especially in the tropics—a condition with which our own service is not unacquainted in the Philippines.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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A Common Misconception of Astigmatic Refraction.—SUTER (*Annals of Ophthalmology*, January, 1905) calls attention to a misconception which we suppose almost every student of refraction has at some time held, and from which many never free themselves. The error is the more widespread from the fact that some text-books actually teach it, and others do not make the matter as clear as it should be. The error is this: that in astigmatism the meridians intermediate between the two principal meridians have each a separate focus, which is situated upon the optic axis somewhere between the focus for the meridian of least curvature and the focus for the meridian of greatest curvature. As a matter of fact there are but two foci upon the principal axis—one for the meridian of least curvature, and one for that of greatest curvature.

The focal lines are two lines perpendicular to the axis at each of the foci, anterior and posterior; the directions of the two lines are at right angles to each other, being parallel respectively to the principal meridian to which each belongs. A pencil of parallel rays refracted by an astigmatic surface is divided into two pencils, one of which is brought to a focus at a point in the anterior focal line, and the other at a point in the posterior focal line. Thus of two rays impinging upon opposite extremities of an intermediate meridian, neither will ever meet the optic axis nor will the two rays meet each other, and, consequently, there can be no focus for rays lying in this meridian.

The Operative Treatment of High Myopia.—FROST (*Brit. Med. Jour.*, November 12, 1904) details his experience and conclusions based upon operations on 39 eyes in 35 patients during the past nine years. The patients' ages ranged from ten to forty-eight years; 2 were lost by suppuration, rather strangely the only cases under twelve years of age. Detachment of the retina occurred in 8 cases, but it is difficult to say that this event was due in every case to the operation; thus in 1 case the detachment did not occur until six years after the operation; in another the interval was four years; in 2 two years; in 3 others it was eight months, four and two months respectively, and in 1 ten weeks. The result was vitiated in several other cases by chronic disease, such as cyclitis, glaucoma, and choroiditis. As regards the degree of myopia, the writer's view is that the operation is of little benefit if the resulting ametropia exceeds 4 D. This condition is only fulfilled if the myopia is not less than is corrected by a lens of —15 D. placed 10 mm. in

front of the eye. This represents a myopia of only 13 D. measured from the cornea.

The optical effect of removing the lens increases with the degree of myopia; an emmetropic eye rendered aphakic requires about 10.50, whereas an eye myopic 25 D. becomes nearly emmetropic; that is, such an eye would have to be 31.1 mm. in length, and this is the posterior focal distance of the cornea.

The visual acuity also must be taken into account when considering the probable benefit to be derived from the operation. The improvement to be expected therefrom can only be due to increased size of the retinal image, so that when the defective vision before operation is due to fundus changes it is irremediable by the operation. At the same time the changes actually visible with the ophthalmoscope afford a very unsafe guide in forming an opinion as to the improvement that may be obtained after the operation. The writer tests the visual acuity by the ability to read No. 1 Jaeger at the patient's far point. A rough but practically useful test.

The operation is usually called for only in young adults, or at the age of puberty; high myopia in young children is very rare. Young eyes thus affected are probably unsound and bad subjects for operation. Older people do not frequently require the operation, because they have chosen their path in life and have become accustomed to the inconvenience of wearing strong glasses. In the series reported only 5 were over thirty-five years old.

Should both eyes be operated on? When improvement has resulted from the operation on one eye it is rare that the same result is to be expected from the other. There is usually a difference of several dioptres in the myopia of the two eyes. Moreover, operation on the second eye deprives the patient of the ability to see near objects without glasses.

In the discussion which followed this paper, which was read before the section of Ophthalmology of the British Medical Association, Dr. E. Landolt remarked that highly myopic eyes were almost always diseased eyes affected with chronic choroiditis and its multiple consequences. Operation on the weaker of the two eyes would probably not yield a great advantage. Operation on the better eye might lead to entire loss of useful sight. Extraction was admissible only on one of two relatively good eyes of about 20 D. myopia.

Posterior Cortical Cataract of Traumatic Origin.—KOLLER (*Annals of Ophth.*, January, 1905) reports the case of a young man who received a perforating flap wound in the upper outer quadrant of the cornea of equitriangular shape, each side about 3 mm. long. Examination six days later showed that the flap was well adapted. In the region of the posterior pole of the lens a delicate star-like, somewhat diffuse opacity was present. Three and one-half weeks after the injury, the opacity of the posterior pole was smaller but much better defined. Vision was $\frac{6}{12}$; with the stenopœic slit it was $\frac{6}{9}$. A better scrutiny of the anterior part of the lens with the ophthalmoscope revealed a condition which had most likely existed from the start, but had escaped notice. In the anterior capsule of the lens exactly corresponding to the inner angle of the corneal wound was an opacity not larger than the finest dot, and adjoining it, outward and downward, the most

anterior layer of the lens showed a delicate, frost-like, bean-shaped opacity, its axis slanting from the nasal to the temporal side. It is now five years since the accident and both opacities are practically unchanged, except that the anterior cortical opacity is larger. The vision with the proper correction is $\frac{5}{5}$.

Posterior cortical cataract of traumatic origin appears to be very rare; perhaps this is only apparent, its presence being hidden behind anterior opacities which are so common after perforating injuries to the lens or contusions of the eye.

Posterior polar cataract is to be distinguished from posterior cortical cataract (Fuchs). Posterior polar cataract is a small white dot at the posterior pole outside the lens; it is congenital and due to incomplete involution of the hyaloid artery. Posterior cortical cataract is much larger and shows the structure of the posterior cortex. It is very common in degenerative affections of the deeper structures of the eye, but has been recorded only a very few times as a sequel to traumatism, piercing wound of the lens, or simple contusion of the eye. It is an interesting and puzzling fact that in the case of perforating wounds of the lens the location of the lens wound seems to have no direct relation to the posterior cortical opacity. The opacity develops shortly after the injury and may result in complete cataract. It generally remains stationary after clearing up somewhat in the first weeks. This clearing makes it likely that the cause of the opacity is not destruction or alteration of the lens fibres, but it may be due to some deposit between them which can again disappear.

Various hypotheses have been advanced to explain this curious formation. Fuchs thinks that it is due to a gorging of preformed spaces (lymph spaces) by a liquid. Some observers have supposed that they had demonstrated the existence of such interfibrillar spaces, but recent researches are against the assumption that preformed lymph spaces exist in the normal lens. Zur Nedden assumes that the traumatism leads to momentary displacement of the lens within the capsule with loosening of the lens fibres at the posterior capsule. In consequence of this loosening some fluid from the vitreous enters into the cementing substance between the fibres of the lens. This hypothesis does not explain why this loosening should take place at the very pole of the lens, and why it should be so absolutely symmetrical. Further experiment is necessary to explain this curious and interesting phenomenon of the lens reacting with a change at its posterior pole to injuries inflicted on its anterior surface.

An Epidemic of Acute Catarrhal Conjunctivitis Due to the Koch-Weeks' Bacillus.—SNELL, Sheffield (*Lancet*, August, 13, 1904), reports the occurrence of such an epidemic in a rural district embracing about 12,000 inhabitants. Over 200 individuals were affected. The overwhelming majority occurred in children; in all cases both eyes were affected. The cornea was involved in but a single eye in a woman, ending in perfect recovery. Isolation and appropriate treatment soon put a stop to the outbreak.

Congenital Word-blindness.—HINSHELWOOD, Glasgow (*The Ophthalmoscope*, October, 1904), reports another case, making his fifth of this rare condition. The subject was a boy, aged twelve years, who had

the greatest difficulty in learning to read. While he was unusually bright at figures and an average speller, he could rarely read by sight more than two or three consecutive words, but came to a standstill every second or third word, and was unable to proceed unless he were allowed to spell the word aloud, thus appealing to his auditory memory, or so spell it silently with his lips, thus appealing to his memory of speech movements. His refraction, visual acuity, and fundus were normal.

This defect appears to be due to a difficulty in acquiring and storing up in the brain the visual memories of words and letters. It may not extend beyond this visual group of images. The visual memories of numbers and musical notes may be quite normal. These facts are to be explained by the different groups of visual images being stored up in different, but probably contiguous, areas, most likely the angular gyrus of the left side. As defect of the gyri on both sides is rare, re-education can be accomplished with great perseverance. The obstacle is probably due to the fact that all the centres involved in speech are on the same side of the brain.

Glaucoma and the Glaucoma Theories.—PATTERSON (*Scottish Medical and Surgical Journal*, July, 1904) gives an interesting exposition of the present views upon this subject. The source of the aqueous humor is to be found in the epithelium of the ciliary region. Leber describes the secretion of the aqueous as a transudation or filtration process depending upon a difference of pressure between the blood in the vessels and the fluids in the chambers of the eye. Doubtless, however, the epithelial cells play more than a passing or mechanical role. No direct nervous mechanism has been demonstrated. By acting indirectly through the vasomotor nerves, the balance between blood pressure and intraocular pressure, and, consequently the amount and character of the secretion may be greatly modified. Normal aqueous strongly resembles normal saline solution. After puncture of the anterior chamber it contains much albumin and fibrin and is spontaneously coagulable. The vitreous humor is really a tissue of the finest fibriles, containing in their meshes a fluid almost identical in composition with the aqueous. This fluid can pass through the zonula, but the interchange of fluid in the meshes of the vitreous is probably very slow.

Almost the only path for the elimination of fluid from the eye, at least in man, is the filtration angle. In addition, there is probably some absorption of fluid by the vessels of the iris and a slow passage from the vitreous by the optic nerve. Schlemm's canal is a venous rather than a lymph sinus. It often contains blood corpuscles. It communicates by the minutest pores with the anterior chamber; the anterior ciliary veins have direct communication with it, so that fluid from the anterior chamber passes directly into the veins of the sclerocorneal margin.

The fluid contents of the eyeball stand at a pressure midway between the arteriocapillary and venous blood pressures. It equals about 25 mm. Hg.; physiological variations from this standard are possible only in a very limited range. The tension is the same in the aqueous and vitreous chambers. This is not altered by accommodation. The intraocular tension can never exceed the blood pressure in the ciliary arteries, 90 to 110 mm. Hg. The blood pressure in the capillaries of the ciliary region has been estimated at about 50 mm. Hg.; that in the veinlets into which the fluid filters from the anterior chamber at about 10 to 15

mm. Hg. Mydriatics and myotics have no influence on the tension of the normal eye. Increase and diminution of the blood pressure have a corresponding effect respectively upon intraocular tension. Division of the sympathetic appears to cause some transient diminution of tension.

Glaucoma. Attempts at producing glaucoma in animals have usually succeeded but imperfectly. The most successful was Bentzen, in Leber's laboratory, who produced permanent increased tension by wounding the tissues at the filtration angle, which caused adhesions, shutting of the angle. In secondary glaucoma an evident mechanical obstruction can usually be demonstrated, giving rise to retention.—*e. g.*, fragments of lens matter in traumatic cataract, blocking the spaces of Fontana, etc.

In primary glaucoma these mechanical explanations no longer suffice. A great obstacle in the way of investigation is the difficulty of obtaining glaucomatous eyes for examination in the earlier stages of the disease, of which very few cases have been published. The later lesions are (1) cupping of the disk with accompanying optic atrophy, (2) closure of the filtration angle, (3) degenerative changes in the intraocular bloodvessels. Most of the glaucoma theories stand in close relation to 2 or 3. The older views of glaucoma associated with the names of von Graefe and Donders were based on the idea of hypersecretion. These views have been largely abandoned. A new conception of glaucoma was introduced by Knies. This regards the increased tension as caused by retention due to adhesions of the root of the iris to the cornea, blocking the filtration angle. Weber explained this by forward pressure of the swollen ciliary processes. While the angle is undoubtedly frequently blocked, most observers no longer regard this as a primary lesion, having been found absent in certain glaucomatous eyes examined in the early stage. Czermak and Birnbacher have laid special stress on the diseases of the vertex veins, endophlebitis, and periphlebitis, but this does not appear to be constant. Priestley Smith brought forward a theory which considers the circumlental space as becoming narrowed, especially in hypermetropic eyes, from the growth of the lens. There are many difficulties in the way of accepting this. Panas appears to favor a modified secretion theory due to changes in the nerves and bloodvessels. Abadie's idea that glaucoma is due to disease of the sympathetic has not found much favor.

The author, like most observers, favors a retention theory. One great point in its favor is the well-known fact that glaucomatous eyes remain hard for a long time after enucleation. There is practically no outflow from such eyes. The extreme shallowness of the anterior chamber in glaucoma has never been satisfactorily explained. Injection of fluid into the vitreous in animals' eyes causes very little shallowing of the anterior chamber. In eyes liable to acute glaucoma a very shallow anterior chamber is the rule. With the blocking of the filtration angle, there is probably an advance of the root of the iris and anterior part of the ciliary body, which further increase the shallowness of the anterior chamber.

In explaining the more chronic forms of glaucoma, the writer would lay most stress on changes in the vascular system, both general and local. Signs of vascular degeneration, both local and general, are most marked in the variety which occurs in association with retinal hemorrhage. Being characteristically a disease of advanced life, its relation to changes in the vascular system seems especially noteworthy. Zimmer-

man, in a recent paper, expresses the view that glaucoma depends on a reduction of arterial blood pressure leading to nutritive changes, transudation, etc.; but experiment and clinical evidence seem alike opposed to this opinion. In fact, the writer believes that such patients present an increased rather than a lower arterial pressure.

It does not seem worth while to study further the changes in glaucomatous eyes unless the examination be made in the earlier stages, perhaps such opportunities might be found in animals, as the disease is said to occur frequently in aged dogs kept under highly artificial conditions.

PATHOLOGY AND BACTERIOLOGY.

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The Importance of Bacillus Pneumonia Friedlaender as a Cause of Pneumonia.—STÜHLERN (*Centr. f. Bak. und Parasitknd.*, 1904, Bd. xxxvi., p. 493) records ten cases of pneumonia in which the bacillus pneumoniæ Friedländer was recovered either from the consolidated lung at autopsy or from the sputum during life. In three instances this bacillus was found in the lung unassociated with other organisms. In five cases there was a mixed infection of bacillus pneumoniæ and diplococcus lanceolatus, and twice these two organisms, together with staphylococcus pyogenes aureus, were cultivated from the sputum. At autopsy the lungs presented the picture of an atypical lobar or pseudolobar pneumonia, often hemorrhagic in character. The symptomatology and physical signs were those of an atypical lobar pneumonia, bloody, slimy sputum being common. The prognosis is very unfavorable and the mortality high.

Tuberculosis of the Mitral Valve and of the Aorta.—Tuberculosis of the heart valves may arise in one of three ways: by direct infection through a deposit of tubercle bacilli upon the endocardium, through hæmatogenous infection by way of the bloodvessels of the valves, and from secondary infection of vegetations or thrombi. WITTE (*Ziegler's Beiträge*, 1904, Bd. xxxvi., p. 192), from a review of the literature, concludes that true tuberculous endocarditis is of extremely rare occurrence, and, eliminating many doubtful cases reported, he would include in the above group only the cases reported by Benda, Burkhardt, and possibly the one recorded by Tripier. The author describes a tuberculous infection of the mitral valve following a right tuberculous coxitis and accompanied by generalized tuberculosis. There was no chronic valvular disease, no vegetation, and no mixed

infection. The valve was the seat of a conglomerate tubercle having a caseous centre, in which tubercle bacilli were found. The tubercle was situated within the tissue of the valve. Excluding the possibility of a deposit of bacilli upon the endocardium or an extension of the process from the heart muscle, the author concludes that the bacilli reached the valve by way of the vasa vasorum. Besides the above case, two instances of tuberculosis of the aorta are recorded, illustrating two different modes of infection. In one case there was a miliary tuberculosis of the intima associated with generalized miliary tuberculosis. A few small sclerotic areas were seen through the length of the aorta, and it is assumed that one of these offered a point for infection. In the second case the tuberculous process extended from some neighboring lymph nodes through the wall of the vessel toward the lumen. In neither instance could the origin of the generalized tuberculosis be traced to the lesions in the aorta.

Report on Trypanosoma and Trypanosomiasis, with Special Reference to Surra in the Philippine Islands.—MUSGRAVE and CLEGG (*Report of Superintendent of Government Laboratories in the Philippine Islands*, year ending September 1, 1903) contribute an elaborate and excellently illustrated article on the subject of trypanosomiasis. From their work they conclude that trypanosomiasis is a general infection caused by trypanosoma. The term "trypanosomiasis" in a general sense is used to designate all varieties of the infection as found in different animals. A long list of vernacular names now in use, except surra, should be discarded or else allowed to fall simply as synonyms, save in those cases where the infecting parasite is shown to be a species distinct from Trypanosoma evansi. The disease is distributed over large areas of the tropical and subtropical world, corresponding closely in its dissemination to the malarial zone. Trypanosoma are discussed in general with reference to history, methods of study, general characteristics, including modes of multiplication, agglutination and involution forms, distribution in the body and outside of the body, and the life-cycle of the parasites. The life-cycle is as yet unknown, but it is believed to be acted out entirely within the animal economy. A tentative classification is adopted for purposes of study, and each trypanosoma of importance is discussed with reference to its principal characteristics, habitat, and pathogenesis. Differential diagnosis of trypanosoma of mammals, like the life-cycle, is left an open question, but the authors believe, from a review of the literature and from their own observations, that at least three of the species to which separate names have been given are in reality identical with Trypanosoma evansi. The authors draw attention to the spread of the disease through wounded surfaces in which biting insects, particularly flies and fleas, serve as the principal agents. The prevalence of the disease is dependent upon the presence of a host for trypanosoma and the insects for the transmission of the parasites. Animals which serve as hosts for the perpetuation of the disease during the dry season differ in different countries. In Manila sick horses exist in sufficient numbers to carry the infection from one rainy season to another, but it is thought that cows and rats may also aid in its perpetuation. Statements concerning the infection of pastures and water and the transmission of the trypanosoma through sound mucous membrane have nothing to support them. The path-

ology, anatomy, and symptomatology of the infection in various species of animals are discussed. The manner in which the symptoms vary in different animals makes this discussion necessary in order to come to satisfactory conclusions concerning the diagnosis and intelligently to control the epidemics.

A chapter is devoted to the consideration of the identity and individuality of surra, nagana, dourine, and mal de caderas. As in the case of the parasite, the authors have left the subject open, but they are strongly inclined to believe that these diseases are the same, in which case surra would be the only vernacular name allowable. They find nothing in the clinical study of these diseases to differentiate them, and the only real arguments in favor of their individuality are based upon morphologic differences in the parasites. These differences appear so slight that a positive classification could not be founded upon them.

The study of prophylaxis includes the consideration of quarantine laws intended to prevent the infection and reinfection of the country, as the case may be, and the methods for the control and eradication of the disease in territories in which it already has a foothold. In discussing this matter the writers have limited themselves almost entirely to the consideration of means adapted for destroying the hosts and supplemented by those suitable for combating the carrying agents. Prophylactic and curative serumtherapy have thus far failed to give successful results, but if recent reports from Africa are to be trusted preventive inoculation is not entirely without promise of success. All methods tried for the treatment of the disease have been without results, particular importance, or significance.

The Pathogenesis of the Crisis in Lobar Pneumonia.—Tchistowitch, in the *Annales de l'Institut Pasteur*, 1904, May 25, No. 5, continues his work upon pneumococcic infection. He has performed a series of experiments to determine the principal factors involved in the production of the crisis in lobar pneumonia.

In former researches the author has already established that if animals are subjected to a pneumococcic infection of non-fatal severity a leukocytosis and a very energetic destruction of the pneumococci by the phagocytes take place, while, on the other hand, in fatal infections caused by virulent pneumococci the number of phagocytes in the blood diminishes, and phagocytosis is not apparent. Further work has convinced the author that the crisis is not due to bactericidal properties which have been developed in the body, as he has been able to obtain, even after the crisis, living and virulent pneumococci by puncture of the diseased lung. He has also been able to show that the blood of patients suffering with pneumonia during or even after the crisis does not possess bactericidal properties for the pneumococci procured from the same patient. Rejecting the findings of Isaeff, that the blood of a recovering pneumonia patient possesses active antitoxic properties, the author considers that phagocytosis is the principal factor in the process of recovery. He considers the crisis to be produced by the destruction in the lungs of the majority of the pneumococci by phagocytes. The phagocytosis assumes a more active character in these cases, he thinks, by the appearance in the blood of the stimulins. The

agglutinins, antitoxins, and the other substances of defence are considered to play only a secondary part and to aid in the defence of the organism against pneumococcic infections.

Histological Studies of Xanthoma.—Various theories have been advanced in regard to the nature of xanthoma. The tumor has been considered to be derived from atypical and irregular multiplication of sebaceous cells analogous to the carcinoma development of other glands. It has been considered to result from changes in muscle fibres, and has also been thought to be of endothelial origin. The histologic study of xanthoma by McFARLAND and McCONNELL (*Journal of Medical Research*, July, 1904, vol. xii., p. 69) has led them to believe the tumor to be of connective-tissue origin. They found the chief changes in the reticular layer of the cutis, which consisted of an invasion of its tissue by cells penetrating in an irregular manner, resembling the invasion of the alveolar tissue by carcinoma cells. These cells resemble exactly those of the sebaceous glands, and contain a large number of fat-droplets in their cytoplasm. The authors believe these xanthoma cells, as they have been called, to be formed by a very gradual conversion of connective tissue similar to the normal transformation of ordinary connective tissue into adipose tissue.

Amœbæ: Their Cultivation and Etiological Significance.—MUSGRAVE and CLEGG (*Report from Bureau of Government Laboratories, Biological Laboratory*, No. 18, 1904) in an interesting article described their method of cultivating amœbæ and the results of a series of animal experiments. A stock medium is prepared in the following manner: agar, 20.000; sodium chloride, 0.300 to 0.500; extract of beef, 0.300 to 0.500.

This is made of 1 per cent. alkaline to phenolphthalein. The medium is prepared in the same way as ordinary sugar, then poured into Petri dishes, and upon the surface is inoculated the material containing amœbæ. So far the amœbæ have only grown in the presence of bacteria. No pure cultures were ever obtained; and in order to procure successful cultivations the importance of this bacterial symbiosis must be taken into account. Usually, in cases of amœbic dysentery, or of "amœbiasis," as the authors have termed this disease, growths of at least one type of bacteria present in the intestines aid the multiplication of the amœbæ. The organisms show a decided selectiveness, however, and when transferred to a plate smeared with a mixed culture of bacteria may pick out a single type. Different growths of amœbæ select different bacteria. Thus, by the use of twelve different selected bacteriæ, growth was obtained in 30 per cent. of one series of cases, when the control inoculations made on the same medium without bacteria showed only 2 per cent. of positive results. Amœbæ multiply rapidly in from twenty-four to forty-eight hours at room temperature, moving over the surface of the plate. Separate amœbæ could be transferred from one plate to another, so that a single species could be cultivated and studied. In cultures the parasites about the margin of the growth are most actively motile, and frequently multiplication by fission is observed. In older cultures encysted forms are common.

The wide distribution of amœbæ has always been recognized, and the authors have succeeded in cultivating them, not only from the

excreta, intestinal lesions, and liver abscesses in man, but also from 100 samples of water taken from the hydrants in different parts of Manila, from the washings of vegetables, from a number of fruits and various organic matter. In general, it may be said that the whole surface flora of the Philippine Islands carries a large number of these parasites.

Regarding the significance of the presence of these parasites in the intestinal tract, the authors are inclined to believe, though this fact is not yet proven, that all amœbæ are pathogenic or may become so. Further, that they may produce typical lesions in the intestinal tract of man. By feeding cultures of amœbæ from the stools of individuals suffering from dysentery to monkeys, and in one instance to a human being, diarrhœa was produced, fatal to monkeys. In the intestinal discharges the parasites were found, and in monkeys ulcers were seen in the colon. A very important point from a sanitary view was the fact that the disease could be produced by feeding monkeys with cultures of amœbæ obtained from water or from the washings of vegetables.

Vaccinia and its Parasite (Plasmodium Vaccinæ).—Bosc (*Centralb. f. Bakt. u. Parasitenkund*, Orig., 1904, Bd. xxxvii. pp. 39 and 195), from an extensive study of the skin lesions of vaccinia, concludes that this affection is a true inflammatory disease of neoplastic type ("bryocytose") showing rapid evolution and accompanied by the production of immunity toward the sixth day. The disease is due to the development, at first localized, later generalized, of a true parasite, intracellular and belonging to the class of protozoa, the plasmodium vaccinæ.

The Differentiation of Bacillus Diphtheriæ, Bacillus Xerosis, and Bacillus Pseudodiphtheriæ by Fermentation Tests in the Serum-water Media of Hiss.—Knapp (*Journal of Medical Research*, 1904, vol. xii. p. 475) from a study of 27 cultures of diphtheria bacilli, 10 cultures of xerosis bacilli, and 4 cultures of pseudodiphtheria bacilli concludes that these organisms may be differentiated rapidly and with great ease, according to their behavior upon certain of the sugars. The xerosis bacillus ferments saccharose, while the true diphtheria bacillus does not. Dextrine, on the other hand, is fermented by diphtheria bacillus, but not by the xerosis bacillus. The pseudodiphtheria bacillus differs from both the true diphtheria bacillus and bacillus xerosis, in that it does not ferment any of the sugars tested. Dextrose, mannite, and maltose are all fermented by both the diphtheria bacilli and xerosis bacilli, so that these sugars do not serve to separate the two organisms.

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